CONGENITAL DISLOCATION OF THE HIP

The Results of Conservative Treatment

JOHN WILKINSON and CEDRIC CARTER, LONDON, ENGLAND

From the Orthopaedic Department, the Hospital for Sick Children, Great Ormond Street, London, and the Medical Research Council's Clinical Genetics Research Unit

The aim of this study is to assess the early results of conservative treatment of congenital dislocation of the hip and to draw attention to the factors that influence the results. Recently some authorities have suggested that conservative treatment will be abandoned, because it has failed to produce the required results, and that it will be replaced by routine operative treatment (Trevor 1957, Somerville and Scott 1957). We believe that, whereas conservative treatment is seldom successful in children with bilateral dislocation, it can be successful, as judged by early results, in most patients with unilateral dislocation. The main object of this study was to determine whether in unilateral cases the degree of acetabular slope of the opposite hip gave a reliable indication of the prognosis for the dislocated hip. The effect of other factors such as fragmentation of the femoral epiphysis and persistent eccentric reduction was also studied.

MATERIAL

Over 200 patients with congenital dislocation of the hip who attended the Hospital for Sick Children between 1948 and 1954 were reviewed, but of these only 149, seventeen boys and 132 girls, were assessed. Those not included were a group of patients in whom initial reduction was not successful; a group in whom the dislocation was secondary to arthrogryposis multiplex, spina bifida or septic arthritis; and a group for whom the first radiographs were not available or were unsuitable for measurement. There were 107 patients with unilateral dislocation, ninety-four girls and thirteen boys, and forty-two patients with bilateral dislocation, thirty-eight girls and four boys. One hundred and forty-one patients began treatment in the first three years of life; in eight, treatment did not start until the fourth year.

These patients all had conservative treatment, but in a little under a half (fifty-one unilateral and twenty-four bilateral cases) reduction was by manipulation under anaesthesia, and in the others (fifty-six unilateral and eighteen bilateral cases) the reduction was by fixed skin traction and gradual abduction of both legs. After reduction the two limbs were held in abduction and neutral rotation with either plaster or Denis Browne harness. This position was maintained for nine to twelve months. In the later cases abduction plasters were used for the first two months. Plaster and harness were found to be equally successful in maintaining concentric reduction, and no hip dislocated during the period of splintage when concentric reduction had been achieved. Recurrent dislocation occurred, however, in some instances in which the initial reduction was not complete, and the position of the femoral head was eccentric.

In the later cases there was a preference for the Denis Browne harness because of the greater ease of nursing and the theoretical advantage of favouring development of the hip by permitting more movement. In no case was difficulty encountered in bringing the legs together after nine months in abduction harness.

As the first patient in the series started treatment in 1948 and the last patient started treatment in 1954, the period of follow-up varied between four and ten years. We consider that a three-year follow-up is adequate for assessment of the early results of treatment.

FIG. 1

FIG. 2

Figure 1—Grade 1 result. (Girl aged seven years three months). Figure 2—Grade 2 result. Roof of acetabulum covers only two-thirds of femoral head. (Girl aged eleven years five months.)

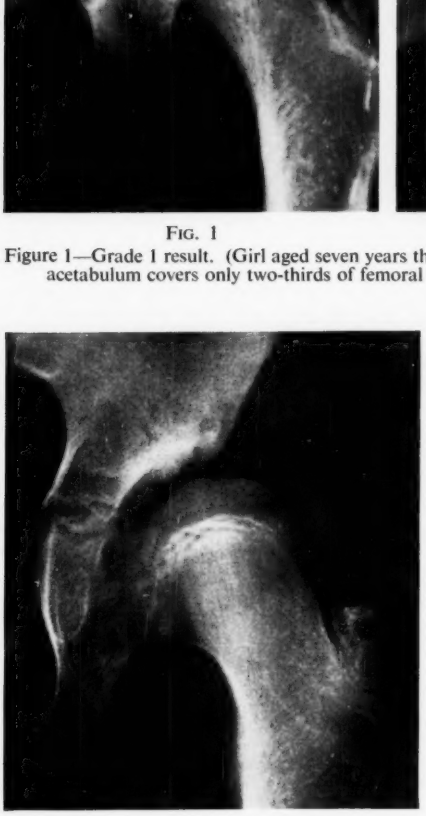


FIG. 3

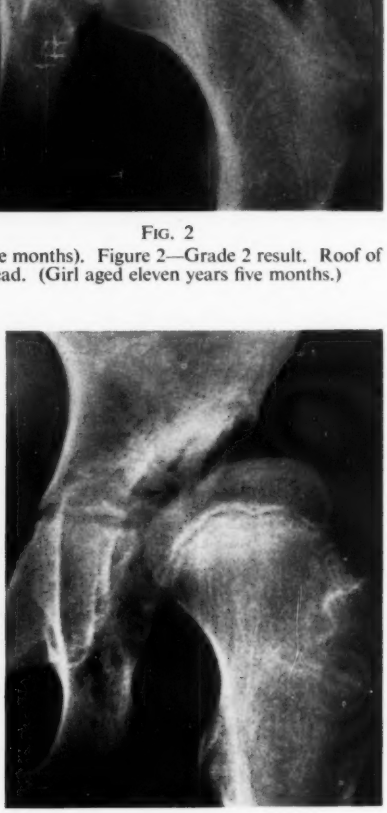


FIG. 4

Figure 3—Grade 3 result. Roof of acetabulum covers less than two-thirds of femoral head. (Girl aged nine years four months.) Figure 4—Grade 4 result. Early subluxation. (Girl aged nine years four months.)

METHODS OF STUDY

Assessment of results—Clinical findings are only a rough guide to the success of treatment. Radiography is, as Somerville and Scott (1957) have emphasised, the most accurate method of assessing the anatomical results. In contrast to reports of the results after open reduction, no patient in this series with a good anatomical result has been handicapped by residual stiffness.

We have graded the result on the radiographs, in some cases supplemented by arthrographs, into five grades. The grading is similar to that used by Severin (1941) and Wiberg (1939), but less detailed and perhaps more practical.

Grade 1—The hip is almost normal. The acetabular roof is well developed and covers at least four-fifths of the femoral head. There is no incongruity of the articular surfaces. Stigmata of the previous dislocation are trivial (Fig. 1).

Grade 2—These hips resemble those in Grade 1, except that there has been a partial failure of acetabular development in that the roof of the acetabulum has grown to cover only two-thirds to four-fifths of the femoral head. There may be no other anatomical abnormality in the hip, or there may be some residual deformity of the femoral head from fragmentation, or there may be persistent eccentric reduction (Fig. 2).

Grade 3—In these hips the acetabular dysplasia is severe and only one-half to two-thirds of the femoral head is covered by the shallow acetabular roof (Wiberg's (1939) C.E. angle = 10 to 5 degrees). Shenton's line is not broken. As with Grade 2 cases, the only other anatomical abnormality in the hip may be incongruity of the articular surface from fragmentation of the femoral head or from persistent eccentric reduction, due usually to soft-tissue obstruction (Fig. 3).

FIG. 5
Grade 5 result. Complete dislocation. (Girl aged eleven years four months.)

Grade 4—In these hips the acetabulum is very shallow and its roof is usually sloping. This may again be an isolated dysplasia, or it may be associated with gross deformity of the femoral epiphysis, secondary to severe fragmentation, or incongruity due to persistent eccentric reduction. Shenton's line is broken, the lateral half being a little above the medial half. Thus, residual subluxation has begun and the hip is unstable. This last factor can be verified with push and pull arthrographs. When a severe degree of fragmentation has occurred, the flattening of the femoral epiphysis is sometimes exaggerated by an associated coxa vara. The C.E. angle is now negative (Fig. 4).

Grade 5—The appearance of these hips is as in Grade 4, but the changes are more severe. The degree of residual subluxation is such that it causes an inch or more of true shortening. In some cases a complete redislocation has occurred. The joint is unstable (Fig. 5).

A number of patients had late surgical treatment when conservative treatment was seen to be failing. These cases are included and have been graded according to the state of the hip before operation. For example, a hip with residual subluxation, Grade 4, after two years of conservative treatment, and then converted by acetabuloplasty to a Grade 3 result, has been kept in Grade 4.

It is considered that these gradings from 1 to 5 reflect the likelihood of future degenerative arthritis in the joints of patients with congenital dislocation of the hip. One cannot be dogmatic about the proportion of patients in each grade who will develop it, since no one has yet followed a series relating the radiological results of treatment, at the age of five to eight years, to those in adult life.

Grade 5 hips, however, undoubtedly are anatomical and functional failures. It is generally accepted that these patients are more susceptible to painful degenerative changes than if they had never been treated. Grade 4 hips may also be regarded as failures. It has been

estimated that about a quarter (Wiberg 1939) or a fifth (Lloyd-Roberts 1955) of cases of osteoarthritis in adults are due to congenital subluxation or dysplasia. Osteoarthritis will be even more prevalent in hips with residual subluxation after treatment, such as we have placed in Grade 4. The future of hips in Grade 3 is less certain, but many may be expected to develop early osteoarthritis. These constitute an intermediate group with an uncertain outcome. The hips placed in Grade 2 and even more those in Grade 1 may be regarded as providing the successes of conservative treatment. Muller and Seddon (1953) reported early symptoms of

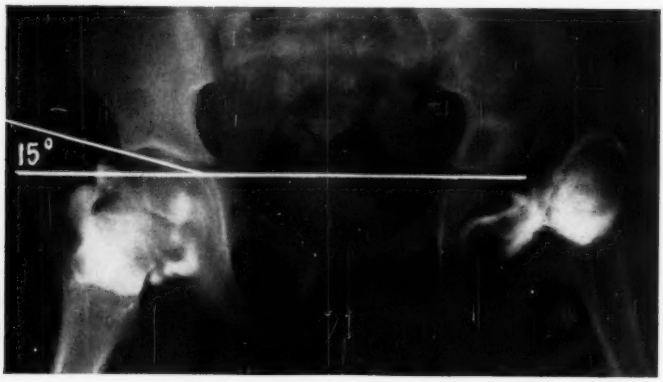


FIG. 6

Measurement of contralateral acetabular angle. A " normal " acetabulum with angle within one standard deviation of the mean for sex and age. (Girl aged two years eleven months.)

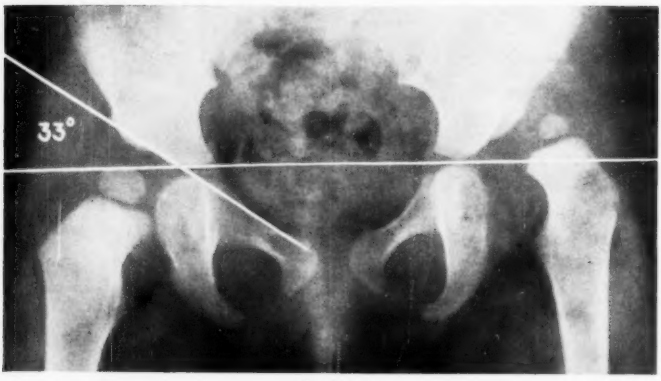


FIG. 7

Measurement of contralateral acetabular angle. A " shallow " acetabulum with angle over two standard deviations above mean for sex and age. (Girl aged one year three months.)

degenerative changes occurring in nearly half the hips that they labelled as " satisfactory " in assessing the early results of conservative treatment. However, many of the hips so labelled would on our classification have been placed in Grade 3. In any case there is no reason to suppose that the hips in Grades 1 and 2 would have been better treated by early operation. It is generally accepted that early operation does not give perfect anatomical results.

Measurement of the degree of acetabular slope in the undislocated hip—It was decided to measure accurately the undislocated hip, to relate the measurement to normal standards, and then to

se. if there were any prognostic significance in the relation to the normal. The measurement ch sen was that of the acetabular slope. This was made by the method first described by Hi genreiner (1925) and later by Severin (1941) (Figs. 6 and 7). When the radiographs in ch dren between six and eighteen months of age were accurately centred the anterior and po erior margins of the acetabular roof were usually superimposed; after this age the two ma gins might not coincide, and in these cases it was our practice to use the more medial (u ally the anterior) margin for measurement. We found it difficult to measure the angle ac rately before the age of six months. For practical purposes this was not important, be use only nine children in this series came to hospital with congenital dislocation of the hi before this age.

Th normal values and variation of the acetabular angle—No mean measurements are known to us for the acetabular slope between the ages of twelve and thirty-five months, the period wi n which we are mostly concerned. (Faber (1937) published figures for normal children at bi h, two and three-quarter months and twelve months; Caffey, Ames, Silverman, Ryder and H ugh (1956) at birth, six months and twelve months. Massie and Howorth (1950) gave

TABLE I

MEAN VALUES AND VARIATION OF ACETABULAR ANGLES FROM SIX TO THIRTY-SIX MONTHS

Age (*months*)	Mean acetabular angle (*degrees*)		Approximate two standard deviation range (*degrees*)
	Girls	Boys	
6	24·7	24·5	±9·1
12	23·0	22·5	±8·7
18	21·2	20·5	±8·4
24	19·4	18·6	±8·0
30	17·7	16·6	±7·6
36	15·9	14·7	±7·2

normal standards from birth to adult life, but unfortunately did not distinguish the sexes.) Some authors have taken a particular angle as the upper limit of normal, without correction for age and sex.

It was considered unjustifiable to take radiographs of normal children to establish mean measurements. Use was made of radiographs of seventy-three girls (146 hips) and sixty-four boys (128 hips) taken over the past seven years at the Hospital for Sick Children for reasons not connected with congenital dislocation of the hip. It is possible that these patients were not altogether normal but it is unlikely that their acetabular angles were to any major degree unrepresentative. Scatter diagrams of the measurements on these radiographs by age and sex are shown in Figures 8 and 9 for girls and boys respectively. The three-monthly means and the regression line of angle measurement to age are shown. The regression line gives the best estimate of the mean value at each age. On either side of it are drawn the lines which are about one and two standard deviations away from it. The mean values and the approximate two standard deviation ranges are shown in Table I.

The distribution of the measurements of the angles is such that about 15 per cent of angles in healthy children would be expected to fall more than one standard deviation above (or below) the mean value for the age and only about 2½ per cent more than two standard deviations above (or below) the mean. These standards will be more accurate when larger

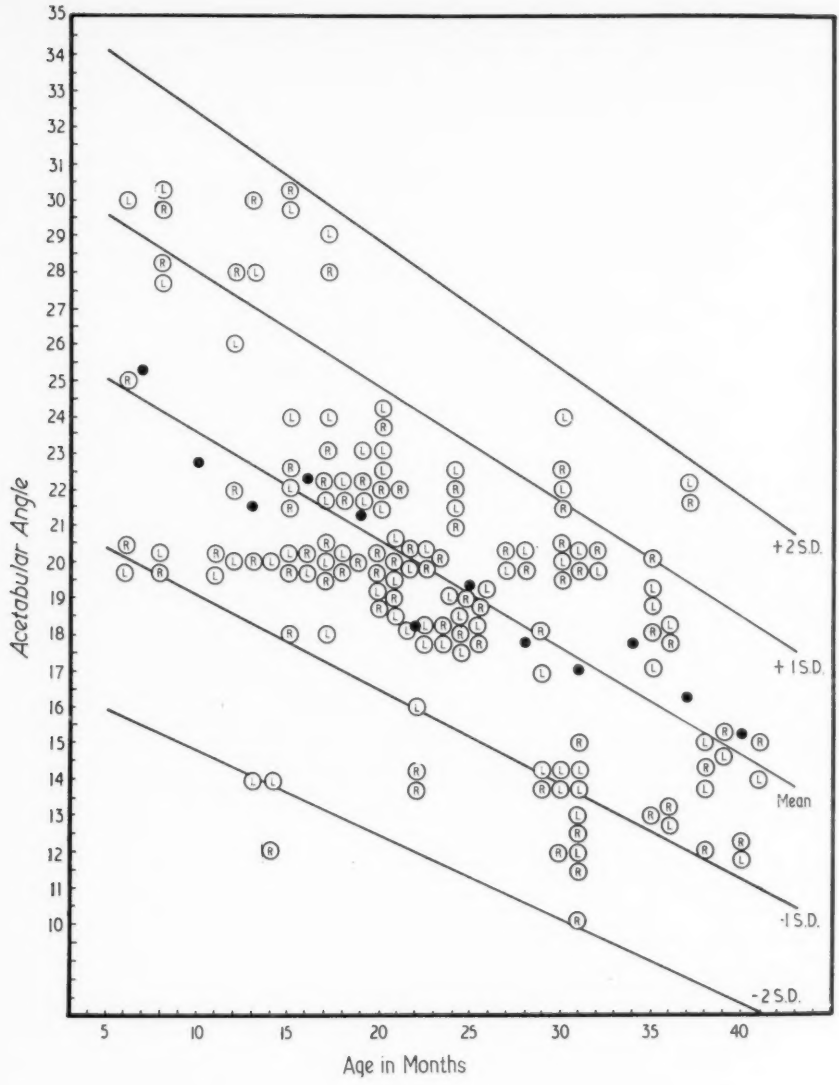


FIG. 8

Scatter diagram of measurements of acetabular angle in normal girls. Three-monthly means and regression line of angle measurement to age are shown. Also shown are the lines indicating one and two standard deviations.

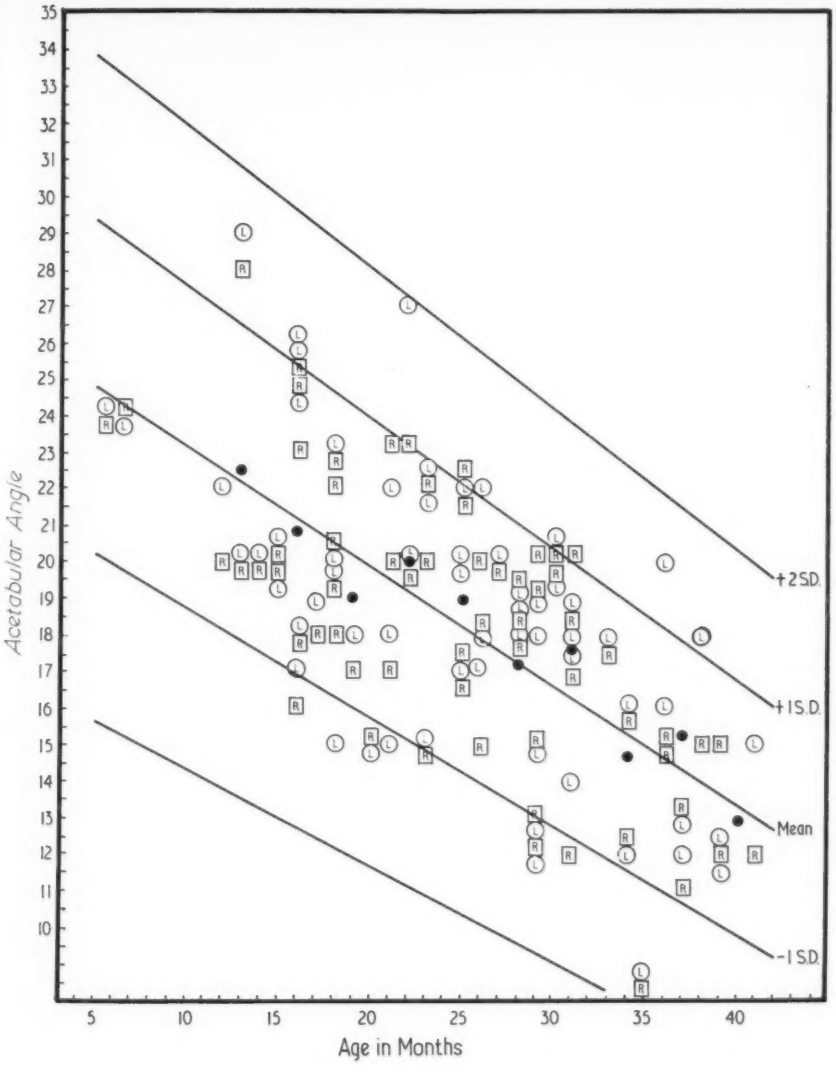


FIG. 9

Scatter diagrams of measurements of acetabular angle in normal boys. Three-monthly means and regression line of angle measurement to age are shown. Also shown are the lines indicating one and two standard deviations.

series of normals become available, and there may be differences in different races. The one-year means in this series (23·0 for girls and 22·5 for boys) may be compared with Faber's (1937) figures from Leipzig (23·6 for girls and 21·3 for boys), and with the figures for whites from New York (21·2 for girls and 19·9 for boys) given by Caffey and his colleagues (1956).

We classified the undislocated hips as " normal," " moderately shallow " and " shallow " according to the size of the acetabular angle. Those with an angle of slope below or within one standard deviation above the mean for sex and age were classed as " normal "; those with angles between one and two standard deviations above the mean as " moderately shallow," and those with angles over two standard deviations above the mean were classed as " shallow."

TABLE II

RESULTS OF CONSERVATIVE TREATMENT OF CONGENITAL DISLOCATION OF THE HIP

Type	Number of hips					
	Result					
	Successful		Intermediate	Failed		Total
	Grade					
	1	2	3	4	5	
Unilateral: girls	32	20	18	8	16	94
	52			24		
Unilateral: boys	3	1	2	4	3	13
	4			7		
Bilateral: girls	5	12	17	20	22	76
	17			42		
Bilateral: boys	2	—	—	2	4	8
	2			6		
Total	42	33	37	34	45	191
	75			79		

THE RESULTS IN GENERAL

The comprehensive results are shown in Table II. In this Table the bilateral cases appear twice, once for each affected hip.

It will be seen that most of the girls with unilateral dislocation were treated successfully, but that there was a considerable proportion of failures. Few boys with unilateral dislocation were treated successfully, but the numbers are small. In the bilateral cases conservative treatment was successful in less than one hip in four.

UNILATERAL DISLOCATIONS:
SIGNIFICANCE OF THE DEGREE OF ACETABULAR SLOPE IN THE UNDISLOCATED HIP

Girls—The relation of the angle of acetabular slope, measured on the radiograph taken at the first visit or—in a few cases—at the first visit after the child was more than five months old, to age and to the result of treatment is shown in Figure 10. It shows clearly the general relation of the degree of slope of the opposite acetabulum to the result of conservative treatment of the affected hip. The findings are summarised in Table III. Treatment was successful in forty-two of the fifty-four girls with " normal " opposite acetabula (Fig. 1).

Acetabular Angle

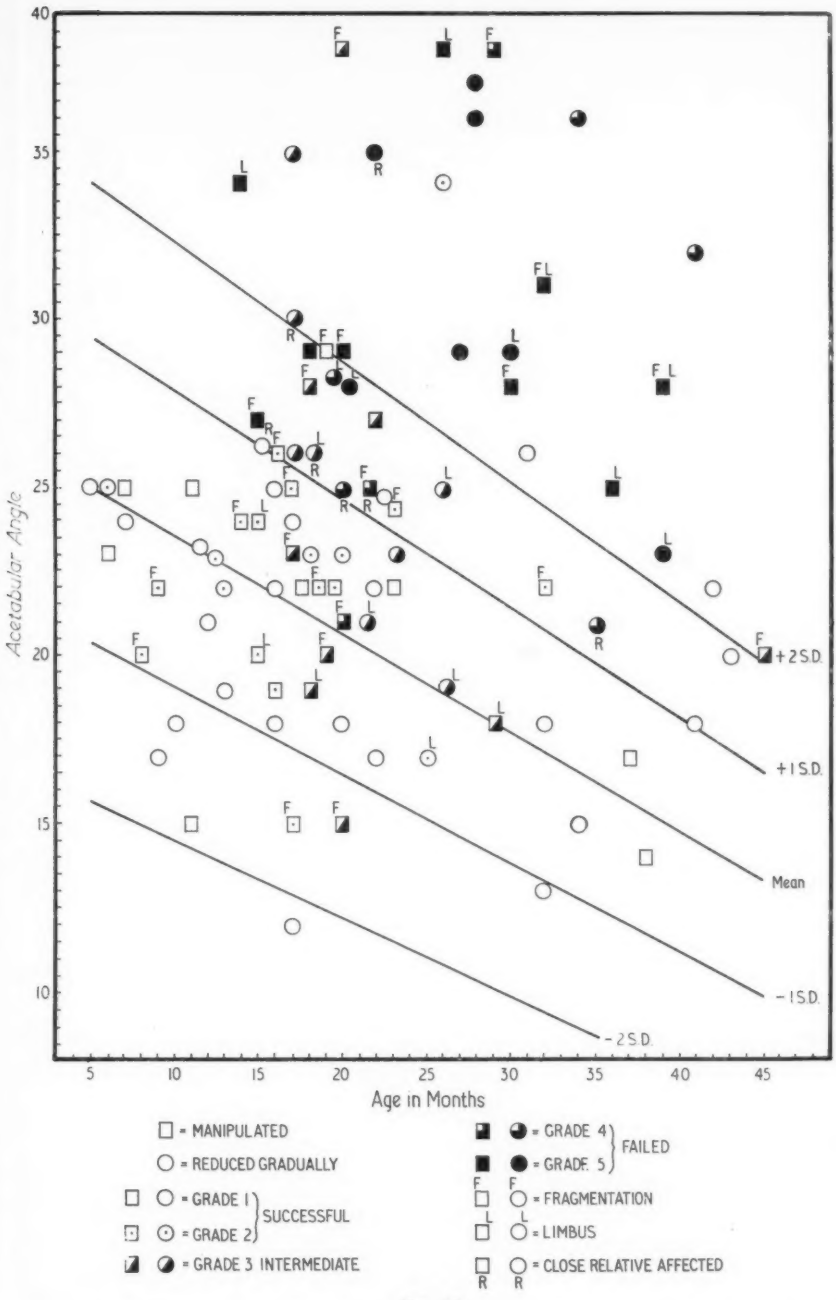


FIG. 10

Girls. The results of conservative treatment related to contralateral acetabular angles in cases of unilateral dislocation. The position on the chart shows the relation of the measurement of the angle in degrees to the mean value for that age in girls. The symbol indicates the type and the result of treatment. The superscript L for limbus indicates the presence of persistent eccentric reduction (due to obstruction by soft tissues—usually but not always an inverted limbus).

It was successful in seven of the nineteen with " moderately shallow " opposite acetabula, and in only three of the twenty-four with " shallow " opposite acetabula (Fig. 12).

The relation of success in treatment to the measurement of the angle on the opposite side may be clarified further if attention is paid to fragmentation of the femoral epiphysis and persistent eccentric reduction. Seven of the eight intermediate results and the one failure, in cases with a " normal " opposite acetabulum, were associated with either fragmentation or persistent eccentric reduction (Fig. 10). Similarly, five of the eight failures with " moderately shallow " opposite acetabula were associated with fragmentation or persistent eccentric

FIG. 11
The result three years after completion of conservative treatment in a girl with a " normal " opposite acetabulum. Grade 1 result.

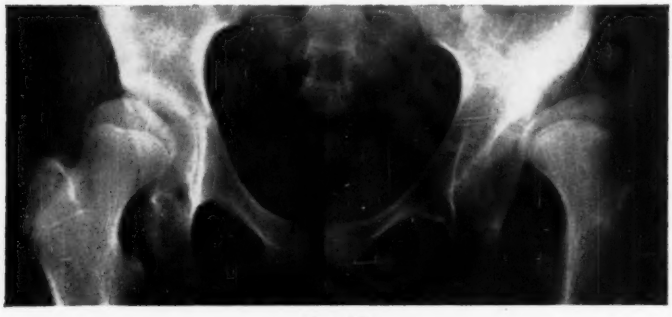


FIG. 12
The result nine years after completion of conservative treatment in a girl with a " shallow " opposite acetabulum. Grade 5 result.

reduction. If cases complicated by fragmentation or persistent eccentric reduction are excluded, conservative treatment was successful in all but one of the thirty-three cases with " normal " opposite acetabula, and the one exception was an intermediate result (Table IV).

Boys—The relationship between the degree of slope on the opposite side and the outcome of treatment in boys is shown in Figure 13. There are fewer observations than in girls and so their significance is less certain. It will be seen that the same general relationship holds as in girls, but that the treatment appears less successful in boys. The main cause of this is probably the high incidence of severe fragmentation. It will be seen that of the eight unilateral cases treated by manipulation only two escaped fragmentation of the femoral epiphysis. All the four failures with angles between the mean and one standard deviation above the mean were associated with severe fragmentation (Table V).

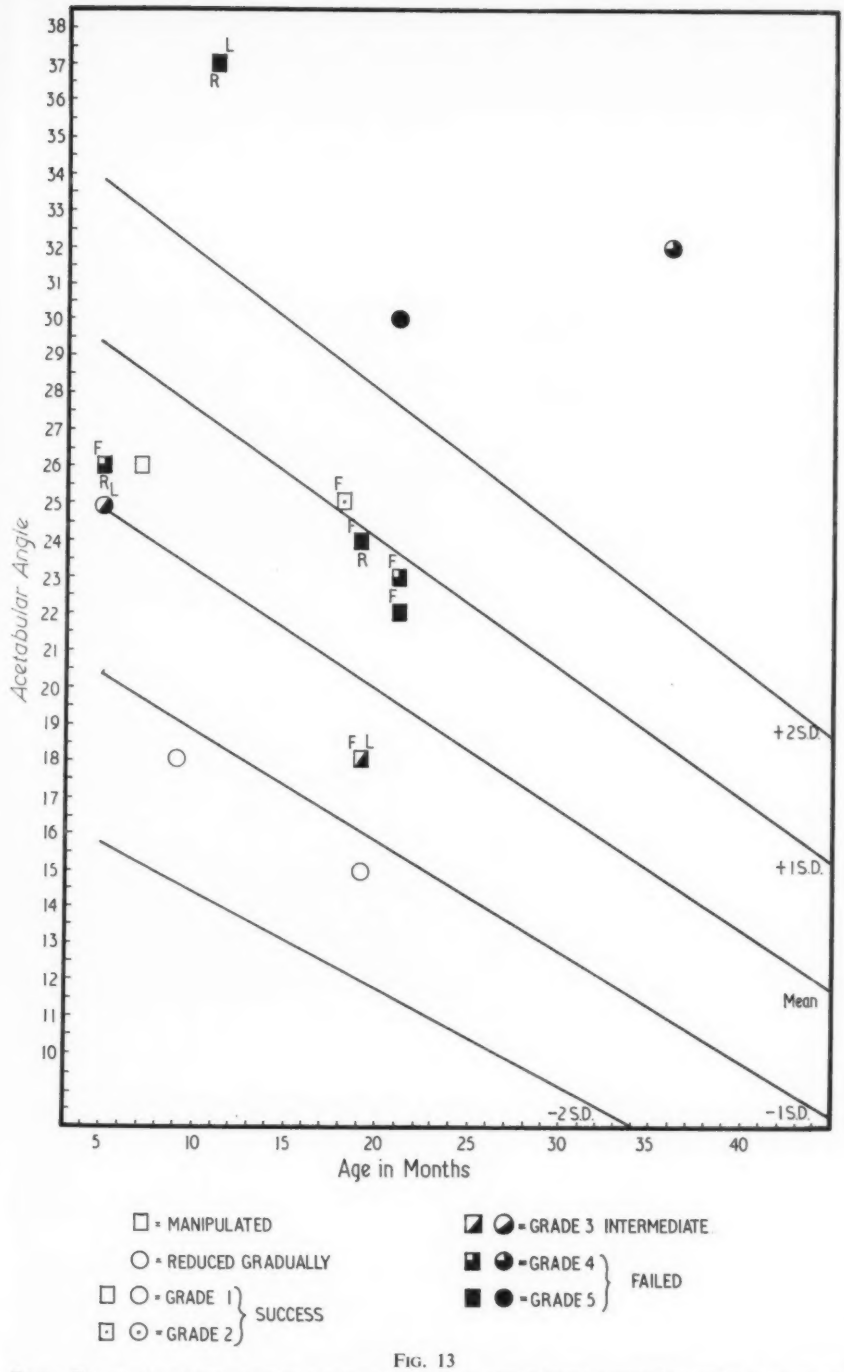


FIG. 13

Boys. The results of conservative treatment related to contralateral acetabular angles in cases of unilateral dislocation. Conventions as in Figure 10.

TABLE III

UNILATERAL CASES: GIRLS

THE RELATIONSHIP OF THE MEASUREMENT OF THE ANGLE ON THE UNAFFECTED SIDE TO THE RESULTS OF CONSERVATIVE TREATMENT

Angle on the unaffected side	Number of hips			Total
	Result			
	Successful	Intermediate	Failed	
	Grade			
	1 and 2	3	4 and 5	
Below the mean	21	3	—	24
Less than one standard deviation above the mean	21	5	1	27
Between one and two standard deviations above the mean	7	5	7	19
More than two standard deviations above the mean	3	5	16	24
Total	52	18	24	94

TABLE IV

UNILATERAL CASES: GIRLS

RESULTS OF CONSERVATIVE TREATMENT, EXCLUDING CASES COMPLICATED BY FRAGMENTATION OR PERSISTENT ECCENTRIC REDUCTION

Angle on the unaffected side	Number of hips			Total
	Result			
	Successful	Intermediate	Failed	
	Grade			
	1 and 2	3	4 and 5	
Below the mean	16	—	—	16
Less than one standard deviation above the mean	16	1	—	17
Between one and two standard deviations above the mean	4	2	3	9
More than two standard deviations above the mean	3	2	6	11
Total	39	5	9	53

TABLE V

UNILATERAL CASES: BOYS

THE RELATIONSHIP OF THE MEASUREMENT OF THE ANGLE ON THE UNAFFECTED SIDE TO THE RESULTS OF CONSERVATIVE TREATMENT

Angle on the unaffected side	Number of hips			Total
	Result			
	Successful	Intermediate	Failed	
	Grade			
	1 and 2	3	4 and 5	
Below the mean	2	1	—	3
Less than one standard deviation above the mean	1	1	4	6
Between one and two standard deviations above the mean	1	—	—	1
More than two standard deviations above the mean	—	—	3	3
Total	4	2	7	13

THE BILATERAL CASES

the forty-two patients with bilateral dislocations the results of conservative treatment were on general unsatisfactory (Table II). There were nineteen successes and forty-eight failures in a total of eighty-four hips (the remaining seventeen hips presenting intermediate results). These results are significantly better than the results in unilateral dislocations with "shallow" opposite acetabula, but they are much worse than those in most of the cases of unilateral dislocations in which the opposite hips were "normal" (Table VI).

bilateral cases the results of treatment cannot usefully be related to the measurement of the acetabular angle on the opposite side, which itself has a dislocation. These angles are very large (usually above 40 degrees) and bear no relation to the potential development of

FIG. 14
Bilateral dislocations. (Girl aged two years five months.)

FIG. 15
Same patient as in Figure 14, seven years after conservative treatment. Both hips show Grade 4 results.

either acetabulum. The radiological appearances in a typical bilateral case, before and after treatment, are shown in Figures 14 and 15. It is noteworthy that the results in the two hips of patients with bilateral dislocations are usually much alike, indicating that their potential for development is similar.

SECONDARY FACTORS INFLUENCING THE RESULTS OF CONSERVATIVE TREATMENT OF UNILATERAL DISLOCATIONS

Sex.—It was noted that the few boys with unilateral dislocations did less well than the girls. There were too few boys with bilateral dislocations to provide any information on sex differences.

Fragmentation of the femoral epiphysis—Many authors have described radiological changes in the density and shape of the femoral epiphysis in congenitally dislocated hips undergoing

TABLE VI

COMPARISON OF RESULTS IN BILATERAL AND UNILATERAL CASES. BOTH SEXES

Type	Number of hips			
	Result			
	Successful	Intermediate	Failed	
	Grade			
	1 and 2	3	4 and 5	Total
Bilateral cases	19	17	48	4
Unilateral: more than two standard deviations above the mean	3	5	19	7
Unilateral: less than two standard deviations above the mean	53	15	12	0

TABLE VII

THE EFFECT OF FRAGMENTATION AND PERSISTENT ECCENTRIC REDUCTION ON THE RESULTS OF CONSERVATIVE TREATMENT. BOTH SEXES

Type	Number of hips					
	Result					
	Successful		Intermediate	Failed		Total
	Grade					
	1	2	3	4	5	
Unilateral: less than one standard deviation above mean						
Fragmented	0	6	*4	4	1	*15
Eccentric reduction	0	3	*7	0	0	*10
Remainder	29	7	0	0	0	36
Unilateral: between one and two standard deviations above mean						
Fragmented	0	4	2	1	1	8
Eccentric reduction	0	0	2	1	1	4
Remainder	4	0	2	2	1	9
Unilateral: more than two standard deviations above mean						
Fragmented	0	0	2	1	†3	†6
Eccentric reduction	0	0	0	0	†8	†8
Remainder	2	1	2	3	6	14
Bilateral						
Fragmented	0	0	3	*7	4	*14
Eccentric reduction	0	0	0	*3	6	*9
Remainder	7	12	14	13	16	62
Total						
Fragmented	0	10	*11	*13	†9	**†43
Eccentric reduction	0	3	*9	*4	†15	**†31
Remainder	42	20	18	18	23	121

* Includes one case with both fragmentation and eccentric reduction.
† Includes two cases with both fragmentation and eccentric reduction.

treatment. There is much confusion about the nature, cause and prognosis of these changes. In this series two groups of changes have been observed: 1) The punctate epiphysis. Simple patches of decreased density appear, but there is no deformity of the epiphysis. These changes were twice seen before treatment was even begun and occurred on four occasions in the opposite undislocated and untreated hip. Changes of this degree are transient and do not lead to permanent deformity of the femoral head. We have disregarded such changes in the analysis. A typical sequence of radiological changes is shown in Figures 16 to 18. 2) Fragmentation of the femoral epiphysis, with associated deformity of the epiphysis. This resulted in residual deformity and incongruity of the femoral head; the head became flattened and enlarged. These appearances may follow a disappearance of the epiphysis and its reappearance as a fragmented line. Alternatively, if the epiphysis is not present at the start of treatment, its appearance may be delayed six to nine months. The flattened appearance of the head was exaggerated in many instances by the development of residual coxa vara. A typical sequence of such changes is shown in Figures 19 to 21.

FIG. 16
Punctate epiphysis (no deformity).

FIG. 17

FIG. 18

Figure 17—Transient changes in the femoral head after conservative treatment of unilateral dislocation. (Girl aged two years six months.) Figure 18—The hip five years later, showing Grade 1 result.

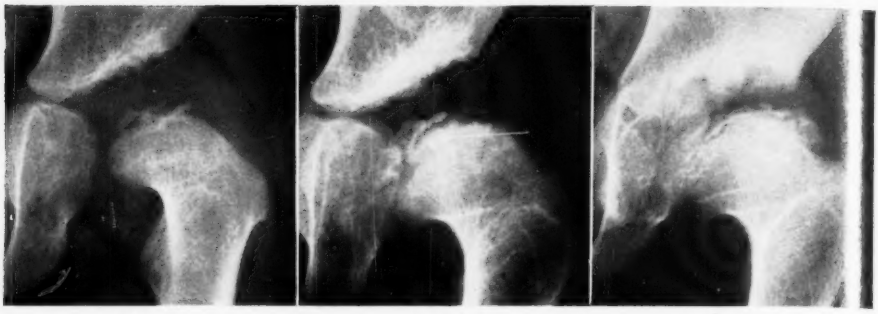


FIG. 19 FIG. 20 FIG. 21

Fragmentation of the femoral epiphysis. Figure 19—The early appearances. (Boy aged one year nine months with a unilateral dislocation treated conservatively.) Figure 20—The appearances at the age of three years two months. Figure 21—The appearances four years later. Grade 3 result. Gross incongruity persists.

Fragmentation was found in twenty-four of the 108 unilateral cases of dislocation and every one of these twenty-four was among the forty-three reduced by manipulation under anaesthesia (Scott 1953). Similarly, fragmentation occurred in fourteen of the eighty four hips in the series of bilateral dislocations and all of these were among the forty-eight hips which were manipulated.

The results in the cases in which fragmentation occurred are shown in Figures 10 and 13, and are compared in Table VII with the cases otherwise similar in which there was no fragmentation. The deleterious effect of fragmentation is perhaps most clearly seen in those unilateral cases in which the results of conservative treatment are usually good—those with a " normal " opposite acetabulum. Instead of the achievement of a Grade 1 result by a majority of cases and Grades 4 or 5 by none, there were no Grade 1 results and five failures among twenty-two cases.

TABLE VIII

ECCENTRIC REDUCTION: BOTH SEXES

Type and treatment	Number of hips		
	Eccentric reduction		Total
	Present early	Persistent	
Unilateral: manipulated	16	9	51
Bilateral: manipulated	7 } 23	6 } 15	48 } 99
Unilateral: gradual reduction	22	10	56
Bilateral: gradual reduction	11 } 33	3 } 13	36 } 92
Total	56	28	191

Concentric reduction—Concentric reduction was achieved early more often when the hip was reduced by manipulation under anaesthesia than when it was reduced by traction and gradual abduction; but the difference is not significant. In over half of the hips in which reduction was at first eccentric, the head of the femur sank in during the first three months of treatment and the final proportion of persistent eccentric reductions was nearly the same in the two groups—about 15 per cent (Table VIII).

Arthrographs were performed in sixteen of the patients with unilateral dislocations reduced by traction and showing eccentric reduction; these all showed an inverted limbus, as described

by Somerville (1953). The arthrographs were repeated in six of these at the time of concentric reduction and all six showed eversion of the limbus. However, eversion of the limbus does not always correct eccentric reduction because in two patients in whom eccentric reduction persisted an arthrograph showed that the limbus had everted.

The effect of persistent eccentric reduction on the results of conservative treatment may be seen from Figures 10 and 13 and Table VI. The effect was similar to, but more severe than, that of fragmentation. The condition caused intermediate results, but no failures, among the unilateral cases in girls with " normal " opposite acetabula. In unilateral cases with " shallow " opposite acetabula, and in bilateral cases, eccentric reduction was invariably associated with failure.

Recurrent dislocation—Recurrent dislocation occurring early in the course of treatment was seen only in patients with persistent eccentric reduction. In the unilateral cases it occurred only when the angle of the opposite hip was also well above the mean. It occurred in every case of persistent eccentric reduction in which the opposite acetabulum was " shallow " (seven girls and one boy). The final result was Grade 5 in all these eight hips.

It is interesting that fragmentation of the femoral epiphysis occurred in only two of the unilateral cases with recurrent dislocation. In the other six there was no fragmentation, in spite of repeated attempts at reduction. This may be because reduction is relatively atraumatic when the hips are shallow.

Of the nine bilateral cases with persistent eccentric reduction, recurrent dislocation occurred in five. Here too the result of treatment was always in Grade 5. Thus, recurrent dislocation was associated with a bad prognosis in both unilateral and bilateral cases.

Contraction of adductor muscles—In five cases of unilateral dislocation, when concentric reduction had been maintained for six months, it was thought that the hip was sufficiently stable to change over to a unilateral hip spica for the last three months and allow mobilisation of the unaffected hip. This was found to be an unwise step as it produced a slight but persistent dysplasia of the dislocated hip. The effect was not sufficient to alter the patient's grading.

Age at first attendance—Figures 10 and 13 show that the age at first attendance had apparently little influence on the results of treatment. On the whole, the patients with large acetabular angles on the unaffected side tended to present later, but the bad results in this group appear to be associated with the large angles rather than with the age at which treatment was begun. When the acetabular angle on the opposite side was below the mean or less than one standard deviation above the mean, the results were good even when the first attendance at hospital was late.

In the group with large angles on the opposite side there is a suggestion that patients brought before the end of the second year have a chance of achieving at least an intermediate result.

Patients with bilateral dislocation as a whole attended hospital for the first time at a rather later age than those with unilateral dislocation, but earlier than those with unilateral dislocation and with a shallow acetabulum on the unaffected side (Table IX).

The relationship between the success of treatment and the age of presentation in the bilateral cases is shown in Table X. There is a significant tendency for the patients attending before the end of the second year to do better than those attending in the first. It should not, however, be assumed that this is due directly to the late attendance. In neither unilateral nor bilateral dislocation is there any evidence to suggest that delay in the start of treatment has a beneficial effect on the early results. Treatment should begin as soon as the diagnosis is made.

A history of a close relative also affected—Only a few patients—seven girls and three boys in the unilateral series (two of the boys were brothers who were both in the series), and one patient (a boy) in the bilateral series—had a parent, brother or sister also affected with congenital dislocation of the hip. The ten unilateral cases are indicated in Figures 10 and 13 by the subscript R.

Conservative treatment in these cases was notably unsuccessful. The results in the unilateral cases were two intermediate and eight failures. Treatment in the bilateral case failed in both hips, making a total of no successes, two intermediate results and ten failures.

In part these bad results are explicable: in one girl and boy because of a " shallow " opposite acetabulum; in two girls because of a " moderately shallow " opposite acetabulum

TABLE IX

AGE AT FIRST ATTENDANCE IN UNILATERAL AND BILATERAL CASES: BOTH SEXES

Type of case	Number of patients		
	Age at first attendance		
	0–11 months	12–23 months	24–48 months
Bilateral cases	3	18	21
Unilateral cases: opposite acetabular angle more than two standard deviations above the mean	1	7	18
Remainder of unilateral cases	16	52	13
Total	20	77	52

TABLE X

RELATION BETWEEN AGE AT FIRST ATTENDANCE AND RESULTS OF TREATMENT: BILATERAL CASES

Age at first attendance	Number of hips					
	Result					Total
	Successful		Intermediate	Failed		
	Grade					
	1	2	3	4	5	
Under 1 year	0	0	0	3	3	6
1–2 years	5	9	8	6	8	36
2–3 years	2	3	9	13	15	42
Total	7	12	17	22	26	84

and fragmentation; in two boys because of severe fragmentation. There is, nevertheless, a suggestion that affection of a close relative is associated with a bad prognosis, independently of the factors we have already discussed.

Anteversion of the femoral neck—The degree of anteversion of the femoral neck did not appear to influence the results of conservative treatment.

DISCUSSION

It is perhaps surprising that other authors have not fully realised the relationship between the measurement of the angle on the opposite side and the success of conservative treatment. The probable explanation is their failure to allow for the normal variation of the angle by age and sex. Severin (1941), for example, taking an angle of 30 degrees as the upper limit of normal after the age of twelve months, concluded that " the hip joints in the unilateral cases of dislocation with dysplasia or subluxation on the ' healthy side ' tend to develop better than those in cases of complete dislocation on both sides, but not as well as the hip joints from

the unilateral cases of dislocation without certain dysplasia or subluxation on the 'healthy' side. This cannot be proved statistically however."

In fact, as we have shown, the relationship between result and angle on the opposite side is a very close one and statistically highly significant. The reason is that the development of this opposite unaffected hip gives, in our opinion, an accurate indication of the potential development of the acetabulum on the affected side. This development on the affected side of the acetabulum, if the dislocation is corrected, may be expected to approach, if not reach, that of the opposite unaffected side. If the acetabulum on the undislocated side is unusually shallow, then it is understandable that the acetabulum on the affected side will not develop sufficiently to give a satisfactory result. In bilateral dislocations the similar results in the two hips indicate a common potential for growth of the acetabulum.

The practical application of these findings is that conservative treatment is the treatment of choice for unilateral cases where the opposite acetabulum is "normal." Fragmentation will not occur if reduction is achieved by traction and gradual abduction.

There is evidence which suggests that the unilateral cases of dislocation with normal opposite acetabula are a distinct group differing from those with unusually shallow acetabula, together with most of the bilateral cases, in their etiology and biological characteristics. This possibility is at present under investigation.

Indications for operation—Two types in the group with "normal" opposite hips require open reduction: those in which eccentric reduction has persisted for more than three months (otherwise there is a risk of an intermediate, Grade 3, result); also the few cases in which the dislocation is not reduced after six weeks of traction.

Early operative treatment is indicated for the unilateral cases with "shallow" opposite acetabula (with angles more than two standard deviations above the mean). It is probably also indicated for those with "moderately shallow" opposite acetabula; there is no way of detecting those in this group that will do well. In both these groups extra-articular acetabuloplasty performed three to six months after reduction would stabilise the femoral head and prevent any tendency to residual subluxation. Such a procedure may not give a Grade 1 result, but is more likely to produce a Grade 2 result than is conservative treatment in these cases.

Early operative treatment is indicated also for the bilateral cases. The results of conservative treatment in this group are worse than for the unilateral cases as a whole, but better than for the unilateral cases with shallow opposite acetabula. There is probably a minority of bilateral cases which are analogous to the unilateral cases with normal hips on the opposite side, and which have a good prognosis if treated conservatively; but there is no way yet of recognising them.

Anteversion of the femoral neck—It has been suggested that femoral anteversion is a cause of residual subluxation or dislocation. In the unilateral cases of this series residual subluxation or dislocation was never seen, except where there was either a shallow opposite acetabulum, persistent eccentric reduction or fragmentation of the femoral epiphysis. If, therefore, anteversion exists it cannot, by itself, be more than an exceptional cause of the failure of conservative treatment. An attempt was made to measure anteversion radiologically in some patients in this series, but these measurements were not considered reliable.

CONCLUSIONS

It is clear that it is possible to select at first attendance, from among unilateral cases of congenital dislocation of the hip, a group of more than half the total in which conservative treatment is the method of choice. The selection is made by measurement of the acetabular angle of the opposite hip, and in this group (provided that concentric reduction is achieved) the results are beyond reproach, so long as epiphysial fragmentation is avoided by discarding any form of manipulative reduction.

In contrast, conservative treatment is at present unsatisfactory for the remaining unil eral dislocations (those with shallow opposite acetabula) and for bilateral dislocations. The erits of the various surgical techniques should be judged by their success in these difficult ca s.

SUMMARY

1. The histories of 149 patients, coming to the Hospital for Sick Children within th first three years of life with congenital dislocation of the hip (191 dislocated hips), and trea d by conservative methods, have been reviewed.

2. The patients with unilateral dislocations (107) have been divided into three groups, acc ding to the angle of slope of the opposite acetabulum. This angle was measured on th first radiograph and related to the mean value for age and sex.

3. The opposite hip was classed as " normal " if the acetabular angle was below or thin one standard deviation above the mean for sex and age; as " moderately shallow " if was between one and two standard deviations above the mean; and as " shallow " if it wa over two standard deviations above the mean. This grouping was found to have a direct b ring on the results of conservative treatment in unilateral cases. *a*) Those with " normal " op osite acetabula—accounting for most of the unilateral cases—responded well. *b*) Those with " moderately shallow " opposite acetabula responded variably. *c*) The group with " shallow " opposite acetabula usually failed to respond.

4. Most bilateral dislocations behaved as unilateral dislocations with shallow opposite hips.

5. Additional factors influencing the response to conservative treatment—sex, age at first attendance, family history, fragmentation of the femoral epiphysis and eccentric reduction—are discussed.

We wish to thank members of the surgical staff of the Hospital for Sick Children, in particular Mr Denis Browne, Mr G. C. Lloyd-Roberts and Professor A. W. Wilkinson, for access to the records of their patients. We are indebted to Mr G. C. Lloyd-Roberts, Professor A. W. Wilkinson, Mr H. H. Nixon and Dr J. A. Fraser Roberts for their advice and criticisms; and to the Department of Medical Photography at the Hospital for Sick Children, for the illustrations.

REFERENCES

CAFFEY, J., AMES, R., SILVERMAN, W. A., RYDER, C. T., and HOUGH, G. (1956): Contradiction of the Congenital Dysplasia-predislocation Hypothesis of Congenital Dislocation of the Hip Through a Study of the Normal Variation in Acetabular Angle at Successive Periods in Infancy. *Pediatrics*, **17,** 632.

FABER, A. (1937): Das Röntgenbild des Hüftgelenks beim Säugling. *Verhandlungen der Deutschen Orthopädischen Gesellschaft*, **32,** 251.

HILGENREINER, H. (1925): On the Early Diagnosis and Treatment of Congenital Dislocation of the Hip. *Medizinische Klinik*, **21,** 1,385.

LLOYD-ROBERTS, G. C. (1955): Osteoarthritis of the Hip. *Journal of Bone and Joint Surgery*, **37-B,** 8.

MASSIE, W. K., and HOWORTH, M. B. (1950): Congenital Dislocation of the Hip. *Journal of Bone and Joint Surgery*, **32-A,** 519.

MULLER, G. M., and SEDDON, H. J. (1953): Late Results of Treatment of Congenital Dislocation of the Hip. *Journal of Bone and Joint Surgery*, **35-B,** 342.

SCOTT, J. C. (1953): Frame Reduction in Congenital Dislocation of the Hip. *Journal of Bone and Joint Surgery*, **35-B,** 372.

SEVERIN, E. (1941): Contribution to the Knowledge of Congenital Dislocation of the Hip. *Acta Chirurgica Scandinavica*, 84, Supplementum 63.

SOMERVILLE, E. W. (1953): Development of Congenital Dislocation of the Hip. *Journal of Bone and Joint Surgery*, **35-B,** 568.

SOMERVILLE, E. W., and SCOTT, J. C. (1957): The Direct Approach to Congenital Dislocation of the Hip. *Journal of Bone and Joint Surgery*, **39-B,** 623.

TREVOR, D. (1957): Treatment of Congenital Dislocation of the Hip. *Journal of Bone and Joint Surgery*, **39-B,** 611.

WIBERG, G. (1939): Studies on Dysplastic Acetabula and Congenital Subluxation of the Hip Joint. *Acta Chirurgica Scandinavica*, 83, Supplementum 58.

CONGENITAL DISLOCATION OF THE HIP

I. G. MACKENZIE, ABERDEEN, SCOTLAND,

H. J. SEDDON and D. TREVOR, LONDON, ENGLAND

From the Institute of Orthopaedics, Royal National Orthopaedic Hospital, London and Stanmore

In the campaign for better results in the treatment of congenital dislocation of the hip there are two possible lines of attack: earlier diagnosis, and a more rational employment of the methods of treatment at present open to us. After early diagnosis, the importance of which is paramount, our chief problem is still how to make the best use of a number of well known manœuvres, and here there is not yet any agreed policy. This paper is a contribution to a debate that seems likely to continue for some years.

Since 1949 two of the authors (H. J. S. and D. T.) have worked together on the treatment of patients with congenital dislocation of the hip and have come to adopt the policy that will be described. This was based on our experience of seventy-five hips* treated before 1949. These patients did not form a consecutive series and were not looked after exclusively by the authors: for these reasons the results of their treatment are not presented in detail.

This paper is concerned with 137 patients treated between January 1949 and December 1953 and followed up for at least five years: brief reference will also be made to 197 patients treated by us between January 1954 and December 1958. Members of the staff of the Royal National Orthopaedic Hospital and other orthopaedic surgeons have been kind enough to refer their patients with congenital dislocation of the hip to one or other of the two senior authors. To all of these colleagues we are greatly indebted.

On one point we can speak with conviction: closed reduction can give reasonably satisfactory and durable results in a fair proportion of cases. This was shown by Muller and Seddon (1953), who reviewed the results of treatment of patients who attended the Royal National Orthopaedic Hospital during the half century between 1891 and 1941. The primary method of treatment in this series was therefore closed reduction. The two main problems are how to deal with a hip which becomes unsatisfactory after an apparently successful closed reduction, and what to do with the patient in whom closed reduction fails or who is too old for it.

The 1949–53 series—In this series we have excluded only those few patients in whom the dislocation did not conform to the standard pattern, namely those with familial laxity of joints or with arthrogryposis multiplex congenita, who present special problems. There remained 137 patients; three of these could not be traced, leaving 134 available for study.

Fifty-three (39 per cent) of our patients presented special problems and had been referred by other orthopaedic surgeons because of difficulties during treatment.

METHOD OF ASSESSMENT

The results were assessed by the method used by Muller and Seddon (1953) in their review of the late results of treatment of congenital dislocation of the hip. Marks were given for the severity of pain in the joint, the amount of restriction of the patient's activities, the range of movement in the joint, and its radiographic appearance. The method of marking is as follows.

Pain (P)—1) Severe even at rest. 2) Severe on movement; little at rest. 3) Some on movement; none at rest. 4) Occasional discomfort. 5) None.

* ...ty treated conservatively, sixteen open reductions, eighteen rotation osteotomies, twenty acetabular reconstructions, and four capsular arthroplasties.

Function (F)—1) Walks short distances only with difficulty. 2) Walks half a mile with a ma ed dip. 3) Walks two miles with a slight dip. 4) Slight limitation of activities. 5) Can do anytl ng.
Movement (M)—1) Little or none. 2) Less than half normal range; or some fixed defor ty. 3) More than half normal range; no fixed deformity. 4) Slight limitation with no ed deformity. 5) Full range.
Radiographic appearance (X)—1) Dislocation, ankylosis, osteotomy, excision of head and r ck. 2) Marked subluxation or gross deformity. 3) Slight subluxation with much defor ty. 4) Slight subluxation with slight deformity. 5) Almost normal.

A joint that caused no pain, which did not restrict the patient's activities in any ay, and which exhibited a full range of movement and nearly normal radiographic appearar es, would thus score 20 points. A unilateral dislocation, eventually treated by arthrodesis, n ght be graded P5, F4, M0, X1=10=Poor.

The results were classified in four groups:

Excellent	=17 points or more	Satisfactory results.
Good	=14–16 points	
Fair	=11–13 points	Clinical failures.
Poor	=10 points or less	

Hips graded fair or poor are recorded as failures. This method of assessment is sim ler than that of Massie and Howorth (1950) and gives a fair picture of the present condition of the hip.

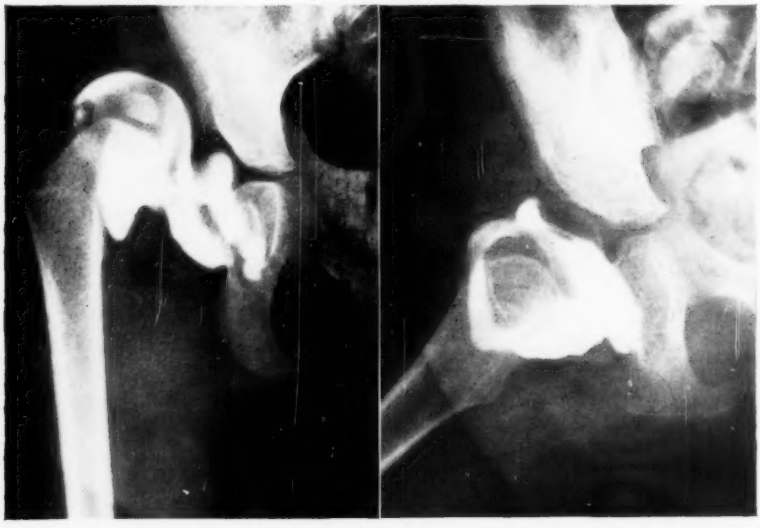


FIG. 1
Arthrographs showing dislocation (*left*) and subluxation (*right*).

GENERAL FEATURES

It has been possible to trace and re-examine 134 of the 137 patients, a follow-up rate of 97·8 per cent. There were 114 girls and twenty boys, giving a sex ratio of 5·7 : 1. The condition was bilateral in thirty-three patients and unilateral in the remaining 101, making a total of 167 hips involved. In 136 of these hips there was a complete dislocation, and in the other thirty-one subluxation only. In a complete dislocation the head loses all contact with the acetabulum, and often soft tissue is interposed between the joint surfaces. In

subluxations the head is in contact with the acetabular rim, and there is seldom any barrier to reduction (Fig. 1).

Platt (1956) questioned the value of Leveuf's distinction between dislocation and subluxation. Although it is, in Platt's words, "no longer a safe guide to treatment" there seems little doubt that the difference in prognosis is sufficient to warrant continued use of the terms.

A family history was obtained in only ten patients (7·5 per cent). Other defects were associated with the dislocation in seven patients (5·2 per cent): treatment of the hip in these patients did not present any special difficulties.

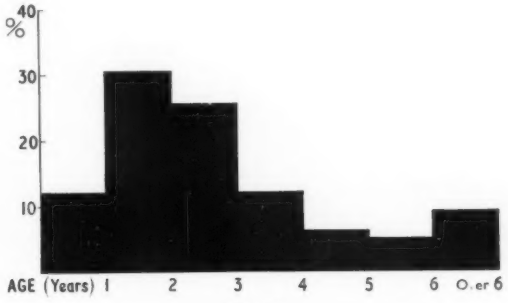


FIG. 2
Age at first attendance.

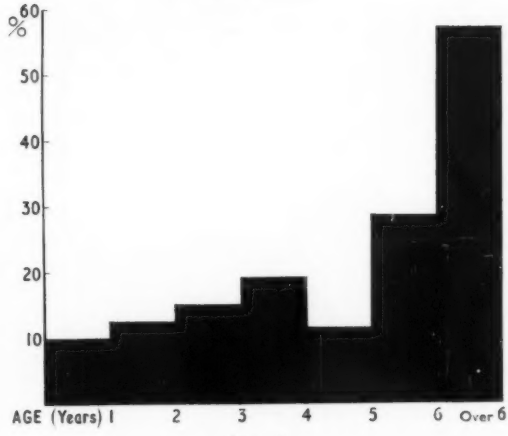


FIG. 3
Percentage of failures at different ages.

Figure 2 shows the percentage of patients attending for treatment at various ages. Too often the diagnosis was made much too late. Figure 3 shows the percentage of failures in the series grouped according to the age of first attendance for treatment. If the child was over five years old when first seen, a third or more of the results were unsatisfactory: under the age of four years less than a fifth were failures.

The results are shown in Table I. The patients have been divided into those who came to us for treatment under the age of three years (ninety-one patients with 114 hips involved), and those who first presented after this age (forty-three patients with fifty-three hips involved).

The results in patients with subluxation were excellent in every patient under three years and in 80 per cent of patients over three. All patients with subluxations who were treated

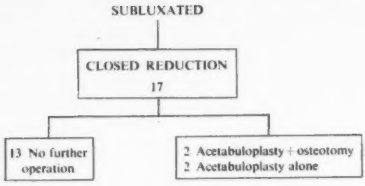


FIG. 4

Figure 4—Method of treatment of subluxations in children under three years of age (seventeen hips).

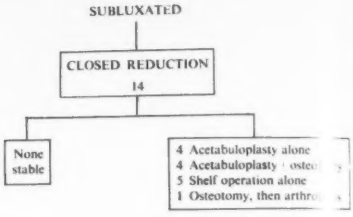


FIG. 5

Figure 5—Method of treatment of sublux ions in children over three years of age (fo teen hips).

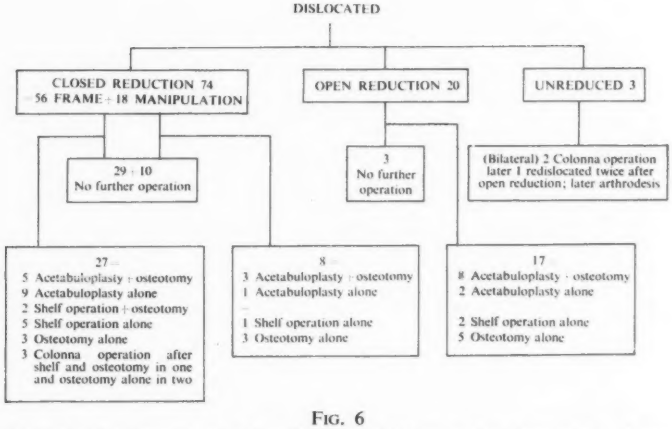


FIG. 6

Method of treatment of dislocations in children under three years of age (ninety-seven hips).

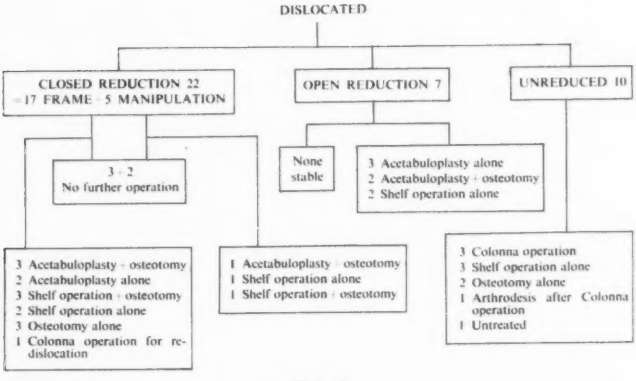


FIG. 7

Method of treatment of dislocations in children over three years of age (thirty-nine hips). All of the ten hips in the " unreduced " category were over five when first seen and most had had several operations elsewhere.

TABLE I

RESULTS IN 134 PATIENTS (167 HIPS)

	Type	Number	Per cent	Excellent	Good	Fair	Poor	Percentage	
				Satisfactory		Unsatisfactory		Satisfactory	Unsatisfactory
Under three years when treatment began									
UNILATERAL	Subluxation	11	6·6	11	—	—	—	100	—
	Dislocation	57	34·2	40	10	4	3	88	12
BILATERAL	Subluxation	6	3·6	6	—	—	—	100	—
	Dislocation	40	24·0	18	11	10	1	72·5	27·5
Over three years when treatment began									
UNILATERAL	Subluxation	10	6·0	3	6	—	1	90	10
	Dislocation	23	13·8	5	9	6	3	61	39
BILATERAL	Subluxation	4	2·4	1	1	2	—	50	50
	Dislocation	16	9·6	6	6	2	2	75	25

after the age of three needed some operation, whereas three-quarters of those under three years required none (Figs. 4 and 5).

Of those with dislocation, 83 per cent had satisfactory results if treatment was begun under the age of three, whereas only 67 per cent had satisfactory results if treatment was started over three: only 8 per cent of the hips in the older group were stable after reduction without further operation, compared with 29 per cent in those under three (Figs. 6 and 7).

INVESTIGATIONS

Arthrography—The enthusiasm that led us to carry out arthrography on 120 hips has waned. Although some elegant pictures (Fig. 1) gave an indication of possible hindrances to reduction we still felt bound to attempt closed reduction first, and in some cases with success in spite of an unpromising arthrograph.

Anteversion—A radiographic method of measuring anteversion (Dunn 1952) was devised by Shelswell in 1950 and was used for some years, but the estimates when checked at operation were sometimes found to be so wide of the mark that it has been abandoned. We now doubt whether estimates of anteversion made before or during the early stages of treatment are helpful in the management of the individual patient. If anteversion is of such a degree that it must be corrected, the need will become apparent during the course of treatment: this is discussed later.

The C.E. angle—This is the angle (Wiberg 1939) between a line drawn in the longitudinal axis of the body through the centre of the femoral head and another line joining this centre to the lateral edge of the acetabular roof. It is used as an index of the development of the acetabular roof and of the stability of the femoral head in its socket. In young children, in whom the acetabular roof is largely cartilaginous it is best measured on an arthrograph done after reduction. Its importance will be discussed later.

TREATMENT

Except in infants, reduction is attempted by gradual abduction on a frame (Scott 1953) with fixed skin traction applied to both legs. When the legs have been abducted so that the angle between them is over 90 degrees, a cross pull is applied through a band round the upper

part of the affected thigh (Fig. 8); the object of this is to coax the femoral head, which is then lying opposite the acetabulum, into the socket; the force used should not exceed three pounds. Clinical signs of reduction are a feeling of resistance deep to the femoral artery and a noticeable improvement in the happiness and comfort of the child. Antero-posterior and lateral radiographs are used to check reduction.

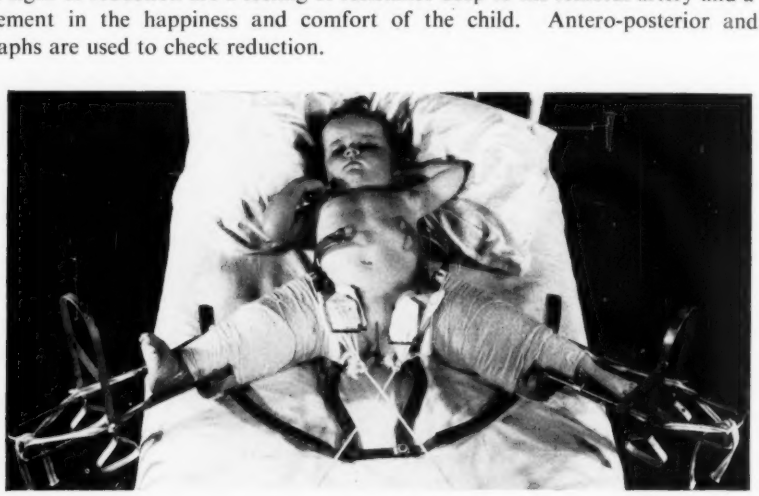


FIG. 8
The abduction frame.

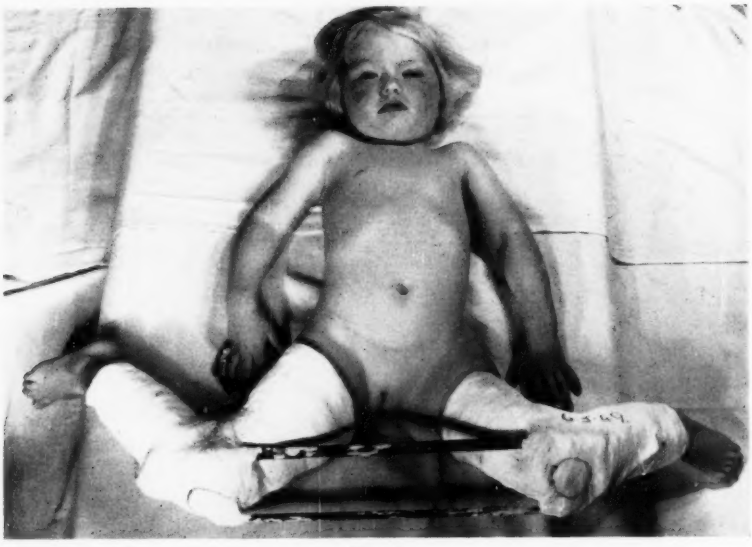


FIG. 9
Batchelor plaster.

If reduction is successful, leg plasters are applied and joined by two crossbars holding the hips abducted and medially rotated (Fig. 9) (Batchelor 1951). These plasters are changed when necessary, usually every three months, and are retained for ten months. The child is then allowed to go free, but is examined frequently to ensure that development of the hip is proceeding normally.

f the radiograph taken when the child has been on the frame for a month shows the fem ral head opposite the acetabulum but standing out (Fig. 10), Batchelor plasters are still app ed and the position is reviewed after three months. Like Severin (1950) we are convinced that a barrier to reduction can sometimes be overcome if the head is held opposite the acet bulum. If the head has not sunk home after three months in a Batchelor plaster the child is a owed to go free, and open reduction is undertaken when the hips are fully mobile.

Not more than about four weeks should be spent in trying to obtain reduction on a frame. Lon er immobilisation may lead to osteoporosis, and we have seen collapse of the femoral hea in such a case at the time of open reduction. For this reason, if reduction has not been obt ned within one month, the child is allowed free, and open reduction is carried out only afte an interval and when the density of the bones has returned to normal.

Open reduction is used if closed reduction fails. After operation the hip is immobilised in a hip spica in the position of greatest stability, which is usually one of medial rotation and alw ys one of abduction. If anteversion is excessive, as shown by the degree of medial rotation req red, rotation osteotomy is carried out about a month later and fixation in plaster is

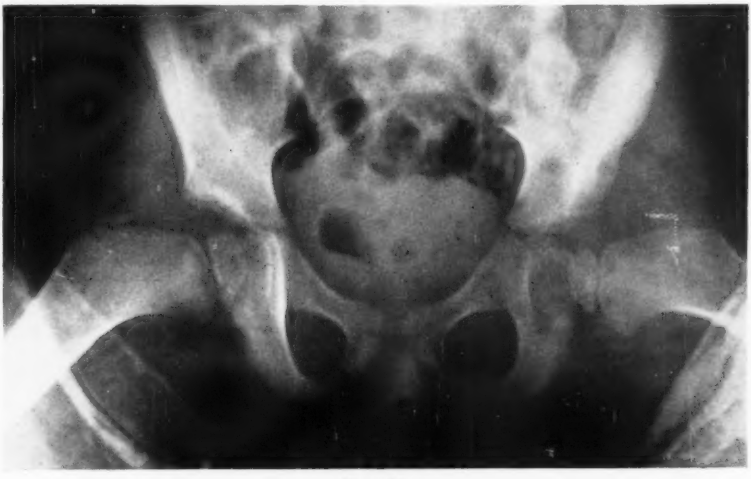


FIG. 10
Head standing out after closed reduction.

continued for a further two months. If the radiograph, in older children, shows very poor development of the acetabular roof, or if, at open reduction, the cartilaginous roof is seen to be sloping, acetabuloplasty is carried out at the time of open reduction.

Closed reduction—Subluxations present no problem in reduction (Figs. 4 and 5) and the following discussion is concerned with the treatment of complete dislocation (Table II).

Provided that reduction could be obtained by manipulation the result was eventually satisfactory in 80 per cent of cases. Manipulative reduction had been attempted by other surgeons in some of the cases later referred to us, but we ourselves used it only in infants under six months or in rather older children with subluxations: stability after manipulation was less common than after frame reduction (Table II) and the greatest age at which manipulation was followed by a satisfactory result was four years (Table III). Thus, except in a young infant, we consider that manipulation is inferior to reduction on a frame in a hospital where the use of this apparatus is well understood.

Reduction on a frame was successful in the dislocations of over half the children under three years, but of under half in the older children: Crego (1948) found that stable seating after

TABLE II

DISLOCATION ALONE: METHOD OF REDUCTION

		Frame	Manipulation	Open reduction	Not r	uced
Under three	97 hips (*per cent*)	57·6	18·6	20·7	3	
Stable without further operation	(*per cent total*)	29·9	10·4	3·2	–	
Satisfactory result	(*per cent group*)	83·8	89	70	–	
Over three	39 hips (*per cent*)	44	13	18	2	
Stable without further operation	(*per cent total*)	7·7	5·1	0	–	
Satisfactory result	(*per cent group*)	82·3	80	100	–	
Greatest age for satisfactory result	(*years*)	5	4	6	7 Colo	na

TABLE III

GREATEST AGE FOR SATISFACTORY RESULT

	Age (*years*)
Unilateral subluxation	12
Unilateral dislocation	6
Bilateral subluxation	5
Bilateral dislocation	4
Frame reduction	5
Manipulative reduction	4
Open reduction	6
Avascular necrosis	4
Acetabuloplasty	8
Corrective shelf operation	6
Shelf operation for pain	18
Colonna operation	7

TABLE IV

OBSTRUCTIONS TO CLOSED REDUCTION

	1949–1953 (30 hips)	1954–1958 (62 hips)	Total (92 hips)
Limbus	21	30	51
Acetabular tissue	15	20	35
Mis-shapen femoral head	15	12	27
High medial attachment of inferior capsule	4	19	23
Shortened psoas tendon	6	17	23
Enlarged and/or long ligamentum teres	8	13	21
Hour-glass constriction of capsule	10	7	17

closed reduction occurred in only 55 per cent. In our series the proportion was even smaller: less than one in three of those under three, and less than one in ten of those over three, obtained a satisfactory result without further operation (Table II). The greatest age at which frame reduction was followed by a satisfactory result was five years (Table III). This finding is the same as that of Hass (1951). Provided reduction could be obtained on a frame, the final result was satisfactory in over 80 per cent (Table II).

Open reduction—Open reduction was used only when closed reduction had failed. It was necessary in about one in five hips. Even in a child over four years old it is worth while going through the motions of closed reduction on a frame because the operation is thereby made easier: it might have been more effective if adductor tenotomy had been used. Stability after open reduction alone was rare (Table II): several redislocations occurred through failure to carry out an acetabuloplasty at the time of open reduction. It appears that the acetabulum is often seriously defective in the case requiring open reduction, and acetabuloplasty should always be considered. The greatest age at which open reduction led to a satisfactory result was six (Table III).

The reasons for the failure of closed reduction were noted at the time of open reduction, and the findings in ninety-two hips are shown in Table IV: this includes patients treated since 1953. The infolded limbus was the commonest single cause of difficulty, but of all the interposed tissues it is perhaps the one most likely to be squeezed out of existence by the femoral head. Other factors may be equally important, and more than one obstruction to reduction may be present. In a recent case the limbus had been removed by an experienced surgeon. After some months the hip redislocated. On exploration it was found that a ligamentum teres, which at its femoral attachment looked innocent enough, expanded medially into a tough sheet of tissue which filled about one-third of the acetabular cavity.

Failure to achieve stable concentric reduction, even after attempted open reduction (Figs. 6 and 7), is uncommon under the age of three,* but occurred in one in four of the older patients—a reminder of the penalties of late diagnosis.

Acetabular reconstruction—The most unpredictable feature in the treatment of a dislocated hip is the behaviour of the acetabulum. It is commonly believed that normal development depends on concentric reduction of the femoral head (Massie 1951). Smith (1958) showed that in animals the acetabulum failed to develop after experimental dislocation of the hip. In spina bifida acetabular dysplasia is probably secondary to paralytic dislocation of the hip although a primary defect of both hip and spine cannot be excluded (Carr 1956). On the other hand, Hart (1949) and Hass (1951) believed that acetabular dysplasia was the primary lesion, and this was why well reduced hips sometimes redislocated.

There is probably truth in both views. In the unreduced hip the acetabulum becomes progressively more inadequate. On the other hand good reduction is no guarantee that the acetabulum will develop normally. We have already mentioned that in a hip requiring open reduction the deformity of the acetabulum is often such that acetabuloplasty is called for immediately; even after closed reduction the acetabulum may deteriorate or at least remain inadequate. Massie (1951) thought that the acetabulum passed through a critical period of development between the ages of five and eight years, and we agree that constant vigilance is called for even after an apparently concentric reduction.

In this series, failure of acetabular development was dealt with in two ways. If the bone was likely to be pliable—and it always is under the age of five—an acetabuloplasty was carried out: the existing acetabular roof was levered downwards over the head, and the resulting gap packed with bone wedges from the ilium or pieces of rib from the bone bank. If the bone was expected, or found, to be rigid, a shelf operation was done; a full-thickness graft from the ilium was used to extend the acetabular roof laterally over the head.

* In a unilateral case redislocation occurred twice after two open reductions, and arthrodesis was later carried out. In a bilateral case redislocation occurred after open reduction and was treated by capsular arthroplasty of both hips.

Although it is usual to describe all these reconstructions as shelf operations the distinction we have made is not unprecedented or otiose. In the operation that we call acetabuloplasty the sloping roof is levered down to its proper level; in the other, that we call a shelf operation, the existing roof is merely prolonged laterally. Our French colleagues rightly distinguish between these operations, but whereas the simple lateral extension of the roof is neatly described as a " butée ostéoplastique " there are at least three terms, all clumsy and one inexact, for what we have here described as acetabuloplasty.

FIG. 11
Sclerosis of acetabular roof while still sloping.

These operations fell into two groups, corrective when subluxation had recurred, and preventive when it was thought likely that it would occur, as when a very sloping roof was found at open reduction.

At first we hesitated to employ preventive acetabuloplasty. It is an operation carried out on a child whose hip is clinically normal but shows a radiographic blemish. The operation is not a minor affair and we know of one death. We have also seen delayed healing of the wound and prolonged, though never permanent, limitation of movement of the joint. However, with improvements in anaesthesia and operative techniques our confidence has increased, and we now prefer to carry out acetabuloplasty before subluxation has started—preventive acetabuloplasty. This tendency towards earlier operation is shown in Table V.

There are three radiological features which may help in deciding whether acetabular reconstruction is required. Wiberg (1953) examined the C.E. angle in 200 hips and stated that an angle of under 20 degrees was abnormal. Laurent (1953) found the measurement particularly useful in children over three years. Yet, in fifteen of our forty-three hips successfully treated by closed reduction alone, the C.E. angle was under 20 degrees while the child was in plaster.

Hass (1951) stated that sclerosis of a steeply sloping acetabular roof indicated that regeneration had finished and that operation was indicated (Fig. 11). We have found this a more reliable sign than the C.E. angle, but the absence of sclerosis of a sloping roof is no contra-indication to operation.

Ossa acetabuli may appear in normal hips after the age of eleven years: similar centres may be seen in congenital dislocation as early as two years. Wiberg (1953) thought they indicated a good prognosis, and we confirm this.

The frequency with which we employed these operations for roof reconstruction is shown in Table V. They were required more often in the older children. The greatest age for a successful result after acetabuloplasty for subluxation was twelve. A shelf operation, as here defined, is not a true reconstruction of the acetabulum, and there were no successful cases above the age of six: it was used with decreasing frequency in children (Tables III and V). Like Viberg (1939) and Merle d'Aubigné (unpublished work) we have found that, for the relief of pain in older patients, the operation has a different and more effective place.

TABLE V

ACETABULAR RECONSTRUCTION

Frequency of employment				
1949–1953	**Under 3 (114 hips)**		**Over 3 (53 hips)**	
	Number	**Per cent**	**Number**	**Per cent**
Preventive acetabuloplasty	13	11·4	6	11·3
Corrective acetabuloplasty	19	16·7	12	22·7
Preventive shelf operation	4	3·4	8	15·1
Corrective shelf operation	8	7·0	8	15·1
Total . .	44	38·5	34	64·2
1954–1958	**Under 3 (140 hips)**		**Over 3 (98 hips)**	
	Number	**Per cent**	**Number**	**Per cent**
Preventive acetabuloplasty	43	30·5	12	12·4
Corrective acetabuloplasty	10	7·1	8	8·1
Preventive shelf operation	3	2·1	3	3·0
Corrective shelf operation	0	—	2	2·0
Total . .	56	39·7	25	25·5*
Satisfactory results (1949–1953 only)				
	Number	**Per cent**	**Number**	**Per cent**
Preventive acetabuloplasty	10	77·0	6	100
Corrective acetabuloplasty	17	89·5	10	83·3
Preventive shelf operation	3	75·0	8	100
Corrective shelf operation	5	72·5	7	87·5

* Follow-up in this group is too short to show the numbers of corrective acetabuloplasties and shelf operations that may be required.

The results of these operations are good (Table V). Acetabuloplasty is easier over the age of three, and, because the results of corrective acetabuloplasty are much the same as those of preventive acetabuloplasty, there is no need for haste when a young child has a defective acetabulum.

Rotation osteotomy—The importance of anteversion of the upper end of the femur as a cause of dislocation is still uncertain. Hass (1951) considered that anteversion was unimportant under the age of three. Platou (1953) thought that anything over 30 degrees required correction,

but Shands (1958) showed that the angle may diminish spontaneously after the hip is reduced. Muller and Seddon found that 70 per cent of hips with estimated anteversion of over 45 degrees became subluxated after reduction compared with 47 per cent of those with less than 45 degrees of anteversion.

Laurent (1953) drew attention to an abnormality to which insufficient attention has been paid: frontal inclination of the socket—an acetabular anteversion—which may well be a more important factor. Langenskiöld (1952) also held this view; his remedy for this condition is transplantation of the insertion of iliopsoas to the outer side of the femur.

FIG. 12
Hip requiring varus and rotation osteotomy.

The need for operation to correct anteversion can be determined by taking radiographs with the hips in neutral and in full medial rotation. If the latter view shows the position of the head in the acetabulum to be significantly improved, osteotomy should be carried out. If open reduction of the hip or acetabular reconstruction is carried out, the most stable position of the hip is determined before the wound is closed. The degree of medial rotation will then indicate whether rotation osteotomy is required.

In this series rotation osteotomy was done in twenty-eight hips (24·8 per cent) in children under three, all with anteversion of over 45 degrees; five (18 per cent) of the hips remained unsatisfactory. There were in the same group twenty-seven hips in which osteotomy was not done in spite of an estimated anteversion of over 45 degrees: the failure rate in these was 26 per cent. The difference is not great. In the patients over three rotation osteotomy was done in nineteen hips (36 per cent), all with anteversion of over 45 degrees, and the failure rate was 36·8 per cent. There were ten hips in which osteotomy was not done although anteversion was thought to be over 45 degrees, and the failure rate was 30 per cent.

These figures suggest that the importance of femoral anteversion may have been overstressed in the past. Other factors, notably acetabular anteversion and valgus of the femoral neck may be equally, if not more, important.

Excessive valgus of the femoral neck (Trevor 1958) can be corrected by taking a subtrochanteric wedge based medially, and any anteversion can be corrected at the same operation. Of eighty osteotomies done since 1953 twenty-one were for the correction of valgus as well as rotation. Figure 12 shows such a case: the radiograph is taken in full medial

rotation and shows both femoral heads well seated. On the normal (right) side the outline of the epiphysis is semicircular, and a line perpendicular to the epiphysial plate passes through the lower part of the sacro-iliac joint. On the left side the medial part of the epiphysis is flattened, giving it the appearance of a peaked cap; a line perpendicular to the epiphysial plate passes lateral to the sacro-iliac joint: varus as well as rotation osteotomy is indicated.

Capsular arthroplasty—Capsular arthroplasty was performed on ten hips in this series and on another eighteen since the end of 1953. Colonna (1953) employs his operation in some types of case in which we use open reduction and acetabuloplasty. We have confined capsular arthroplasty to patients who were either too old for direct reduction, or to those in whom a previous operation had failed, or in whom there was acetabular aplasia of such a degree as to preclude open reduction. It was therefore the last resort short of arthrodesis, and indifferent results in such cases should not be allowed to detract from the merits of capsular arthroplasty when carried out in accordance with the strict conditions laid down by its originator.

Colonna stated that ten years is the upper age limit in unilateral and eight years in bilateral cases. Only four of our ten (pre-1953) patients were within this limit. We had one success and three failures; in one there was redislocation, in the other two avascular necrosis with some loss of movement. Yet even these three patients have enjoyed useful function for six or more years and may continue to do so for some time to come.

The arthroplasty was a salvage operation in the six patients over the age limit (two were thirteen at the time) and fair results were obtained in all. We have yet to decide whether this is worth while, but, in the unilateral case, subsequent arthrodesis will certainly be simpler than if nothing had been attempted. In bilateral cases before the age of six one can obtain satisfactory results by open reduction and acetabuloplasty: between six and eight years capsular arthroplasty is probably the operation of choice: over the age of eight the patients' interests are possibly best served by withholding surgery until the hips become painful.

TABLE VI

RESULTS OF CAPSULAR ARTHROPLASTY

	Good	Fair	Poor
1949–1953	1	7	2
1954–1958	4	13	1

Since 1953 we have operated on a further eighteen hips. Four of these have given satisfactory results; there has been one poor result due to redislocation; and the remainder have been fair: avascular necrosis has occurred in three (Table VI).

This bald statement of the results gives an unduly depressing picture. Patients and their parents are pleased with what they regard as good results in the cases that we have graded only as fair.

COMPLICATIONS

Avascular necrosis—Almost a quarter of the children under three, but only 15 per cent of the older ones, developed this complication (Table VII). In all, the radiographs showed irregularity, increased density and often the appearance of fragmentation in the epiphysis. Residual deformity of the femoral head is more common if epiphysial changes first appear after the age of three, and after four years it always impaired the final result. Thus, although the complication is less frequent in the older children, the prognosis is worse.

The incidence was higher in patients treated in a Lorenz plaster than in those immobilised in the Batchelor plasters. From the evidence obtained at open reduction that the most stable position is usually one of medial rotation, the indications for using a "frog" plaster are rare.

Nicholson (1954) showed that the frog-leg position in plaster affects the intravascular pressure, the most sensitive vessels being the profunda femoris and the medial and lateral circumflex femoral arteries. This effect is abolished by adductor tenotomy, and perhaps we should return to the practice of doing this routinely.

Manipulative reduction carries a higher risk of avascular necrosis than does reduction on a frame (Scott 1953) or open reduction. The condition is not uncommon after acetabuloplasty in children under the age of three; and even after a shelf operation, in which the disturbance of the hip itself is minimal, avascular necrosis can occur in children over three. It occurs too, and understandably, after capsular arthroplasty.

TABLE VII
AVASCULAR NECROSIS

	Under 3 (*per cent*)	Over 3 (*per cent*)
Incidence	24·8	15
Failure rate	25	37·5
Frog plaster	37·5	28·6
Batchelor plaster	16·2	12·5
Frame reduction	12·5	11·7
Manipulation	33·3	20
Open reduction	15	10
Acetabuloplasty	31·3	—
Shelf operation	—	10·5
Colonna operation	16·7	25

TABLE VIII
CASES WITH PROLONGED STIFFNESS

Avascular necrosis	8
Shelf operation	4
Colonna operation	2
Open reduction	2
Splintage	1

The duration of the process, judged radiologically, varied from four months to four years but bore no direct relation to the end-result; transitory changes may be followed by considerable permanent deformity. The importance of the condition has certainly been underestimated in the past. Figure 13 shows the radiographic appearance of a hip reduced by manipulation seven months before, and immobilised in lateral rotation. The following four films (Figs. 14 to 17), taken at yearly intervals, show the slow development of the process and the permanent flattening of the head. The more serious degrees of avascular necrosis are accompanied by stiffness of the hip, and when this happens we feel that the condition should be treated like Legg-Calvé-Perthes' disease by relief from weight bearing until revascularisation has occurred.

Stiffness—Prolonged stiffness of the hip occasionally presents a problem. It occurred in seventeen (10·2 per cent) cases. The associated factors are shown in Table VIII. The

comm nest cause is avascular necrosis. The stiffness will normally yield to treatment by tract n followed by exercises in the pool, but occasionally operation is required. One boy who d been treated elsewhere in a Denis Browne splint for sixteen months required Soutter's soft sue release and subtrochanteric osteotomies on both hips before his legs could be brot t together. This adds force to the argument against immobilisation in the frog-leg

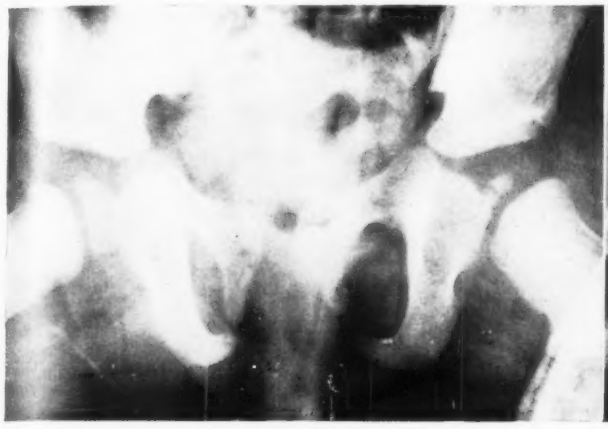


FIG. 13

FIG. 14

FIG. 15

FIG. 16

FIG. 17

FIGS. 13 TO 17

F ure 13—Avascular necrosis affecting the left hip seven months after manipulative reduction of a congenital islocation seven months before. The succeeding Figures (14 to 17) show the progress at yearly intervals.

sition. Stiffness is occasionally due to calcification and ossification in the capsule and ductors of the hip (Fig. 18): it may occur in cases treated conservatively as well as in those erated on, and can affect the normal as well as the dislocated hip. Removal of the ectopic ne gives a disappointingly small increase in the range of movement.

FIG. 18

Ossification in a normal hip after an uneventful reduction of the other hip on a frame, followed by acetabuloplasty.

SUMMARY AND CONCLUSIONS

1. The results of treatment of 134 patients with congenital dislocation of 167 hips are reviewed.

2. Late diagnosis is still a major problem.

3. Subluxations rarely give rise to poor results, but in dislocations first treated over the age of five years there is a one-in-three risk of failure.

4. Manipulative reduction is successful less often than reduction on a frame and carries a higher risk of avascular necrosis.

5. Closed reduction on a frame was satisfactory in 58 per cent of patients under the age of three years, and can succeed up to the age of five.

6. Open reduction was required in 20 per cent of cases under the age of three, and can be used successfully up to the age of six.

7. Seven anatomical barriers to closed reduction have been recognised and two or more are commonly found in one hip when open reduction is performed.

8. The acetabular roof may fail to develop after reduction, especially if this is delayed. A C.E. angle of under 20 degrees does not necessarily forebode this, unless measured on an arthrograph. Sclerosis of a sloping acetabular roof is an indication for operation. Acetabuloplasty is the proper operation for a sloping acetabulum and can be done successfully up to the age of twelve. Over this age, a shelf operation should be performed; this is appropriate also in younger patients in whom the curvature of the acetabulum is normal but does not extend far enough laterally. These operations were required in 38 per cent of hips treated in patients under the age of three, and in 64 per cent over this age. There is a one-in-three risk of avascular necrosis when acetabular reconstruction is done in patients under three.

9. Anteversion, if excessive, should be corrected by subtrochanteric osteotomy, and any valgus of the femoral neck should be corrected simultaneously.

10. Unilateral dislocations in patients over the age of six are best treated by Colonna's operation. In our few bilateral cases over this age our results have been disappointing.

11. Avascular necrosis is less common but more serious when it occurs over the age of three. Manipulative reduction and the use of frog-leg plasters are two avoidable factors which appear

to increase its incidence. The more serious degrees are accompanied by stiffness of the hip, and when this sign is present weight bearing should be avoided.

12. Prolonged, though rarely permanent, limitation of movement occurs in some 10 per cent of cases. In a few, operative correction was required.

REFERENCES

BATCHELOR, J. S. (1951): Symposium on Congenital Dislocation of the Hip. *Journal of Bone and Joint Surgery*, **33–B,** 288.

CARR, T. L. (1956): The Orthopaedic Aspects of One Hundred Cases of Spina Bifida. *Postgraduate Medical Journal*, **32,** 201.

COLONNA, P. C. (1953): Capsular Arthroplasty for Congenital Dislocation of the Hip. *Journal of Bone and Joint Surgery*, **35–A,** 179.

CREGO, C. H., Jun., and SCHWARTZMANN, J. R. (1948): Follow-up Study of the Early Treatment of Congenital Dislocation of the Hip. *Journal of Bone and Joint Surgery*, **30–A,** 428.

DUNN, D. M. (1952): Anteversion of the Neck of the Femur. *Journal of Bone and Joint Surgery*, **34–B,** 181.

HART, V. L. (1949): Congenital Dysplasia of the Hip Joint. *Journal of Bone and Joint Surgery*, **31–A,** 357.

HASS, J. (1951): *Congenital Dislocation of the Hip.* Oxford: Blackwell Scientific Publications Ltd.

LANGENSKIÖLD, F. (1952): On the Transposition of the Ileopsoas-Muscle in Operative Reduction of Congenital Hip-Dislocation. *Acta Orthopaedica Scandinavica*, **22,** 295.

LAURENT, L. E. (1953): Congenital Dislocation of the Hip. *Acta Chirurgica Scandinavica*, Supplementum 179.

MASSIE, W. K. (1951): Vascular Epiphyseal Changes in Congenital Dislocation of the Hip. *Journal of Bone and Joint Surgery*, **33–A,** 284.

MASSIE, W. K., and HOWORTH, M. B. (1950): Congenital Dislocation of the Hip. Part I. Method of Grading Results. *Journal of Bone and Joint Surgery*, **32–A,** 519.

MASSIE, W. K., and HOWORTH, M. B. (1951): Congenital Dislocation of the Hip. Part II. Results of Open Reduction as Seen in Early Adult Period. *Journal of Bone and Joint Surgery*, **33–A,** 171.

MASSIE, W. K., and HOWORTH, M. B. (1951): Congenital Dislocation of the Hip. Part III. Pathogenesis. *Journal of Bone and Joint Surgery*, **33–A,** 190.

MULLER, G. M., and SEDDON, H. J. (1953): Late Results of Treatment of Congenital Dislocation of the Hip. *Journal of Bone and Joint Surgery*, **35–B,** 342.

NICHOLSON, J. T., KOPELL, H. P., and MATTEI, F. A. (1954): Regional Stress Angiography of the Hip. *Journal of Bone and Joint Surgery*, **36–A,** 503.

PLATOU, E. (1953): Rotation Osteotomy in the Treatment of Congenital Dislocation of the Hip. *Journal of Bone and Joint Surgery*, **35–A,** 48.

PLATT, H. (1956): Congenital Dislocation of the Hip: the Role of Open Reduction. In *Modern Trends in Orthopaedics*. Second series. Edited by Sir Harry Platt. London: Butterworth & Co. (Publishers) Ltd.

SCOTT, J. C. (1953): Frame Reduction in Congenital Dislocation of the Hip. *Journal of Bone and Joint Surgery*, **35–B,** 372.

SEVERIN, E. (1950): Congenital Dislocation of the Hip. Development of the Joint after Closed Reduction. *Journal of Bone and Joint Surgery*, **32–A,** 507.

SHANDS, A. R., Jun., and STEELE, M. K. (1958): Torsion of the Femur. *Journal of Bone and Joint Surgery*, **40–A,** 803.

SMITH, W. S., IRETON, R. J., and COLEMAN, C. R. (1958): Sequelae of Experimental Dislocation of a Weight-Bearing Ball-and-Socket Joint in a Young Growing Animal. *Journal of Bone and Joint Surgery*, **40–A,** 1,121.

SOMERVILLE, E. W. (1953): Open Reduction in Congenital Dislocation of the Hip. *Journal of Bone and Joint Surgery*, **35–B,** 363.

SOMERVILLE, E. W. (1953): Development of Congenital Dislocation of the Hip. *Journal of Bone and Joint Surgery*, **35–B,** 568.

SOMERVILLE, E. W., and SCOTT, J. C. (1957): The Direct Approach to Congenital Dislocation of the Hip. *Journal of Bone and Joint Surgery*, **39–B,** 623.

TREVOR, D. (1958): Osteotomy in the Treatment of Congenital Dislocation of the Hip. *Proceedings of the Royal Society of Medicine* (Section of Orthopaedics), **51,** 1,045.

WIBERG, G. (1939): Studies on Dysplastic Acetabula and Congenital Subluxation of the Hip Joint. *Acta Chirurgica Scandinavica*, **83,** Supplementum 58.

WIBERG, G. (1953): Shelf Operation in Congenital Dysplasia of the Acetabulum and in Subluxation and Dislocation of the Hip. *Journal of Bone and Joint Surgery*, **35–A,** 65.

SUPPURATIVE ARTHRITIS OF INFANCY

Some Observations upon Prognosis and Management

G. C. LLOYD-ROBERTS, LONDON, ENGLAND

From the Victoria Hospital for Children, Tite Street (St George's Hospital Group), and the Hospital for Sick Children, Great Ormond Street, London

Some infants with pyogenic arthritis develop alarming radiographic signs which may give a false impression of the extent to which the joint has been damaged by the infection. The prognosis may, in fact, be relatively good and a favourable outcome predicted with confidence in some of these children.

The surgeon who relies upon radiological signs alone and ignores clinical features may in time be embarrassed by a surprisingly good result when he has warned the parents that a serious disability will follow. He may also be misguided enough to operate unnecessarily or, by not operating, he may lose the opportunity to alleviate the effects of the disease. Such mistakes can occur because the decalcification caused by the infection extends beyond the limits of the epiphysis into the metaphysis, where secondary changes may further suggest irreparable damage not only to the epiphysis and metaphysis but also to the intervening epiphysial plate. The final shape of the articulating surfaces is, however, dependent not upon the degree of decalcification of the ossific nucleus or the nearby metaphysis (these changes are reversible), but upon the damage caused to the epiphysial cartilage and plate which cannot be seen in an early radiograph. It was, indeed, demonstrated by Haas (1919) that it is possible to scrape out the entire epiphysial ossific nucleus without affecting the ultimate shape of the bone.

The thirteen patients reported here to illustrate certain features of infantile pyogenic arthritis affecting the knee and hip were all seen within the last three years, so it is not very rare. Several of the cases arose in the practice of colleagues who, knowing my interest, have generously allowed me to treat their patients or to see them, and have made their records available to me. Although it seems likely that prompt treatment by antibiotics aids the survival of the joint it should be noted that an illustration very similar to Figure 24 may be found in Jones and Lovett's text-book of orthopaedics (1923). This patient and three others with hip joint involvement reported by Banks, Krigsten and Compere (1940) illustrate the surprising degree of recovery that can occur without antibiotic or chemotherapeutic drugs.

PYOGENIC ARTHRITIS OF THE KNEE

The syndrome is well illustrated by an account of the first patient in the series in whom an error in management was so shaming that a careful reappraisal established the main clinical and radiological features upon which the diagnosis has been made subsequently.

CASE REPORT

Case 1—A girl aged twenty-one months was admitted under Dr C. W. Kesson with left genu valgum (Figs. 1 and 2). At the age of five days septicaemia from umbilical sepsis had been diagnosed and treated with antibiotics. A month later both knees were found to be swollen, thick pus was aspirated and staphylococcus aureus was grown. The organism was insensitive to penicillin but sensitive to erythromycin, which was prescribed immediately and continued for six weeks. Traction was maintained for a further two weeks, and she was discharged to out-patient supervision.

FIG. 1

Case 1—The appearances of both knees at the age of twenty-one months. Note the apparent loss of half the epiphyses on both sides, the sloping epiphysial plate and the metaphysial spurs.

FIG. 2

Case 1—Showing the typical metaphysial spurs in the lateral view. The head of the left fibula has been removed.

When she was first admitted there was a 30 degrees genu valgum deformity on th left with slight genu varum on the right. From the radiographic appearances it was ass ned (rather naïvely as it transpired) that the left lateral femoral condyle, epiphysial plate an part of the metaphysis were destroyed and that similar damage had been inflicted upon the ght medial tibial condyle. The metaphysial spurs were interpreted as indicating the point at v ich longitudinal bone growth had ceased.

It was thought that this situation represented the ideal indication for an attem at epiphysial transplantation, and such was our confidence that we began the operatic by removing the upper end of the left fibula with the intention of transplanting it into the ony lower lateral femoral diaphysis which we expected to find. When the knee joint was op ed.

FIG. 3

Case I—Appearances three years after operation. Note the fibular graft in the left femur and evidence of recalcification in the region of the right medial tibial condyle.

however, some consternation was felt when an apparently undamaged lateral femoral condyle was exposed lying within a joint which, apart from some adhesions, appeared to be normal. Fearing nevertheless that the hidden epiphysial plate might have been destroyed the graft was introduced as originally intended.

Figure 3 illustrates the radiological state at the present time, four and a half years after the infection occurred and two and a half years since the operation. There is no shortening, one and a half inches of genu valgum, insignificant lateral instability and 110 degrees of flexion from full extension of both knees.

Comment—Three months after the operation the child was admitted for reassessment, or rather for the assessment that should have been made earlier. This examination revealed the following signs, the significance and reliability of which have been substantially confirmed in six other patients who are briefly described below.

Clinic l signs in relation to the radiological—1. The cartilaginous condyle could be seen and felt in its correct position with the knee flexed to 90 degrees (Case 4, Fig. 4).

2. M ovements were surprisingly free, being from 180 to 70 degrees on the right and 180 to 90 degrees on the left (after operation). Case 4 (Figs. 5 and 6) illustrates this feature

FIG. 4

Palpating the lateral femoral condyle. The radiograph of this patient (Case 4) is shown in Figure 16.

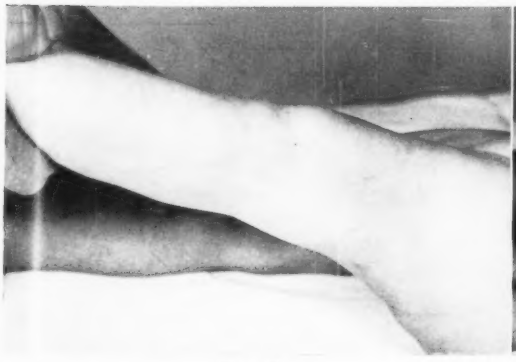


FIG. 5

Figure 5—Shows that full extension is retained.

FIG. 6

Figure 6—Showing the range of flexion. (Case 4; compare Figure 16.)

ever more strikingly. It is inconceivable that such freedom could follow destruction by pus of so large an area of bone within a joint.

3. There was only slight knock-knee deformity (malleolar separation one and a half inches) after two years of seeming loss of the lateral half of the femoral epiphysis. If part of the

FIG. 7
The knee is being abducted in extension. Relative stability is preserved.

epiphysis had in fact been destroyed the d[illegible]rmity would have been much greater. At the pres[illegible] time (over four years since the onset) the deformity [illegible]mains the same.

4. There was (and still is) no shortening. Thi[illegible]ight have been due to bilateral involvement but, e[illegible]n so, shortening has been a problem in only one [illegible]tient (Case 3).

5. The knee in extension was much more stab[illegible] than might have been expected if half of the epiphy[illegible] had indeed been lost (Case 4, Fig. 7).

Radiological signs—1. The metaphysial spu[illegible] are potentially misleading but are in fact good ev[illegible]ence that the epiphysial cartilage is not irreparab[illegible] destroyed. They are present on both sides in this p[illegible]ient and are a constant feature in the radiographs of [illegible] the patients and are illustrated in Figures 1, 2, 10, 1[illegible] 13, 15, 16.

2. In a high quality radiograph of low penetr[illegible]tion the outline of the cartilaginous condyle may be [illegible]een.

3. Arthrographs (Figs. 8 and 9) of the right knee demonstrated the survival of the epiphysis beyond any doubt. There were, however, such obvious signs of this on clinical examination that this was merely a refinement.

FIG. 8

FIG. 9

Case 1—Arthrographs of the right knee showing the outline of the cartilaginous epiphysis of the medial tibial condyle.

OTHER CASES

Short case histories of a further six patients who have had suppurative arthritis of the knee will now be presented and thereafter some salient features will be mentioned.

FIG. 10 FIG. 11

Case 2—Radiographs taken at thirteen months. Note the apparent loss of the medial third of the femoral epiphysis, the sloping epiphysial plate and the metaphysial spur. (Compare Figure 1.) In the lateral radiograph (Fig. 11) the metaphysial spur is well shown. (Compare Figure 2.)

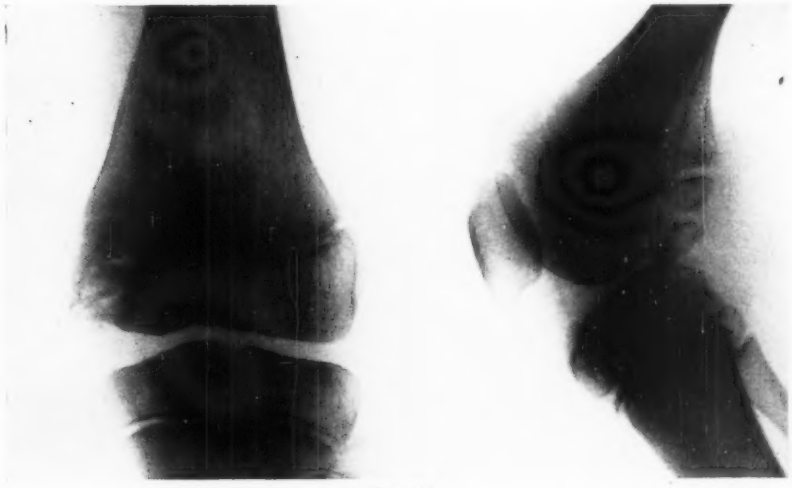


FIG. 12

Case 2—The appearances at thirteen years old. The degree to which the condyle now approaches normal can be seen.

Case 2—A girl, aged thirteen months, was first seen one year after a staphylococcal suppurative arthritis of the knee at three weeks. She had been treated by aspiration, penicillin replacement and systemic penicillin. Radiographs taken at the time of her first attendance are shown in Figures 10 and 11. On examination she had mild genu varum and shortening of half an inch. There was full

knee flexion from a fixed flexion deformity of 15 degrees. She had worn a bow-leg splint for six mo hs but has had no treatment since.

Radiographs twelve years later (Fig. 12) show the remarkable extent to which the lower en of the femur survived the infection. No less remarkable is her clinical state, for from the fully exte led position she has full flexion less 10 degrees, the knee is stable in extension, the limb is less tha an inch short, and genu varum is only just enough to allow a half-inch gap between the extended kn es.

Case 3—An infant boy suffered suppurative arthritis, the pus from which yielded staphyloco us aureus. Two years later the radiograph (Fig. 13) resembled those of Cases 1, 2 and 4. At that me there was no shortening, movements were full and the knee was stable. There was, however, g nu varum of such severity that osteotomy was considered. Nevertheless the deformity gradually impr ed without treatment and was found to be fully corrected four years later. At the same time shorte ng was first noticed: this progressed, so that a discrepancy of two inches existed after a further five y ars at the age of eleven. The opposite femoral epiphyses were accordingly stapled and some improve nt was noted six months later. The boy is now twelve and his radiograph is reproduced (Fig. 14). He plays all games, has no varus deformity and almost full movement. The knee remains stable.

FIG. 13

FIG. 14

Case 3—Suppurative arthritis of knee. Figure 13—Two years after the onset. (Compare Figures 1, 2, 10, 11.) Figure 14—At twelve years old there has been improvement with growth but the final result is less good than in Case 2 (Fig. 12).

Case 4—A girl, aged five weeks, was admitted with swelling of the knee of one week's duration. Aspirated pus yielded staphylococcus aureus. Radiographs (Fig. 15) already showed marked decalcification, translucency of half the condyle and early metaphysial " spur " formation. Penicillin was injected into the joint and systemically, together with Novobiosin and Nystatin by oral tube. The baby was gravely ill with staphylococcal septicaemia, and after ten days tetracycline was substituted for the other antibiotics and the knee was splinted in extension. This treatment was continued for ten weeks and thereafter a polythene splint was substituted.

Eighteen months after the onset there was full movement at the knee (Figs. 4 and 5), the condyle was palpable (Fig. 3) and the knee was relatively stable (Fig. 6). There was no shortening, genu valgum allowed two inches of malleolar separation and she could stand unaided. Figure 16 shows a radiograph taken at this time.

Case 5—A boy, aged two weeks, was admitted with a staphylococcal septicaemia and osteomyelitis of the left femur with symptoms for one day. Radiographs showed an area of translucence in the lower lateral femoral epiphysis which soon spread to include the metaphysis and the lateral half of

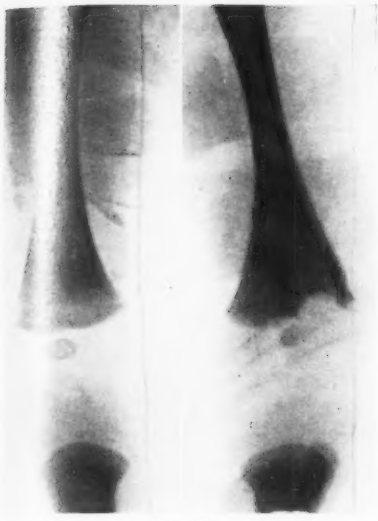


FIG. 15

FIG. 16

Case 4. Figure 15—Radiograph taken seven days after the onset of the infection showing the speed with which decalcification occurs. Figure 16—One year later. The metaphysial spur, sloping epiphysial plate and loss of part of the normally opaque epiphysis are well shown. (Compare Figures 1, 2, 10 and 11.) This was the radiographic picture when the clinical photographs (Figs. 4 to 6) were taken.

the epiphysis. Penicillin and tetracycline were prescribed. A day later an abscess was drained on the lateral side of the thigh and a plaster was applied. Sequestrectomy and curettage of the femur were subsequently performed twice, a further abscess was drained and the child was again admitted for treatment with antibiotics before final healing occurred about three years after the onset.

Seven and a half years later the affected leg was two inches short and genu valgum allowed two and a half inches of malleolar separation. Nevertheless the knee was fully stable and lacked only 10 degrees of flexion. Radiographs showed the lower femoral diaphysis to have remodelled normally: the epiphysial plate was still translucent and there was some bone forming in the lateral femoral epiphysis which was palpable. Further improvement is therefore probable.

Case 6—A boy, aged three weeks, was admitted with a staphylococcal infection of the femur closely resembling that found in Case 5. An abscess required incision, but healing was by primary intention. Systemic treatment was by aureomycin and the organism was again a staphylococcus.

Five years later radiographs showed the typical metaphysial spurs and a translucency in the area normally occupied by the lateral half of the femoral epiphysis. In spite of genu valgum which allowed five inches of malleolar separation the knee was stable and movement full. Shortening was less than half an inch.

Case 7—A boy aged five weeks was admitted with suppurative arthritis of the knee caused by staphylococcus aureus. He was treated by splints and aureomycin for six weeks. Radiographic changes of the type already described affected the lateral half of the distal end of the femur. Full movement, normal longitudinal growth and relative stability were maintained, but in this patient the valgus deformity was of such a degree that supracondylar osteotomy was performed when the child was two years old.

DISCUSSION ON AFFECTION OF THE KNEE

In all seven patients staphylococcus aureus was the organism responsible, and in all fra pus was at one time or another obtained from the region of the knee joint. The de nstrable after-effects were always confined to one half of the femoral or tibial metaphysis an piphysis, and it must be assumed that the vascular supply of the area dictates this.

Cases 2 and 3 are important because they show the recovery that can occur with time. Th hild in Case 2 continued to improve until skeletal maturity; so it is possible that a patient

reported by Hall (1954) at eight years old with stability, no genu varum, 70 degrees of mov ment and two inches of shortening after " loss " at the medial femoral condyle will do lik wise. Case 3 resembles Hall's case very closely and the patient continues to improve.

These patients support the submission that neonatal suppurative arthritis of the nees showing the clinical and radiological features already described have a relatively good pro iosis in terms of mobility and stability. Impairment of longitudinal growth or the developm it of a valgus deformity may occasionally require surgical correction (Cases 3 and 7). We s ould certainly be on guard against over-enthusiastic acceptance of apparently successful transp inta- tion of growing epiphysial plates in such cases as these.

PYOGENIC ARTHRITIS OF THE HIP

Failure to distinguish between decalcification and destruction in pyogenic arthritis f the knee results in no more than unnecessary operation or inaccurate prognosis. A similar m take in the hip, however, may have more serious consequences. It may mislead the surgeo into advising against treatment in the belief that he is dealing with an irreparable dislocation from destruction of the proximal end of the femur when, in fact, enough decalcified bone re ains for repair. I do not infer that destruction never takes place, but merely suggest tha t the apparent absence of bone radiologically does not necessarily imply bone destruction.

The surviving cartilaginous femoral head is sometimes dislocated during the acute phase of the infection (Case 13, Fig. 27), so that a situation develops which is incapable of sponta eous cure whatever the possibilities of remineralisation. These are the patients for whom trea ment is likely to be of benefit. Time may be short, however, because the cartilaginous femoral head does not seem to grow in its ectopic position. This suggestion is supported by findings in two patients operated upon in adult life. These patients had rudimentary cartilaginous femoral heads and necks which were removed in the course of fusion operations. It is perhaps not unreasonable to ask whether these rudiments found in the adult and similar in size to the normal structures in infancy would not have developed more normally had they been replaced in the acetabulum at an early age.

Four of the cases to be presented (Cases 9, 10, 12 and 13) illustrate the advantages of early manipulation or exploration of a hip that is dislocated or " destroyed." In another patient (Case 8) the damage inflicted was found to be irreparable when the hip was explored. No harm came of this and we were satisfied that no opportunity of helping the child had been neglected. This case is not reported in detail below.

CASE REPORTS

Case 9—A girl, aged sixteen days, was admitted with high fever, a swollen thigh and umbilical sepsis. Aspiration of the hip on the same day produced 10 cubic centimetres of pus from which haemolytic streptococci sensitive to penicillin were cultured: the same type of streptococcus was grown from the umbilicus. Penicillin was given on admission and on the next day an abscess which lay in front of the joint capsule was drained. There was no pus in the femoral neck. The wound was sutured and a plaster was applied in abduction. The baby made a rapid recovery and was discharged home after receiving penicillin for six weeks. Pathological dislocation of the hip was then noted (in spite of early abduction) and a divaricator was ordered. This was unsatisfactory; so the plaster was reapplied but was soon removed because the hip was considered to be damaged beyond repair (Fig. 17). Figure 18 shows the appearance of the hip at fifteen months when the child began walking. This was interpreted as showing destruction of the femoral head and the lower half of the neck.

Two years later, when she was three years and three months old, the leg was an inch short and the hip unstable. Arthrography confirmed the dislocation but it suggested that the femoral neck and head were not totally absent (Fig. 19).

At exploration the femoral head was grossly misshapen and resembled a mushroom, but was nevertheless large enough to permit reconstruction of the hip. There was anteversion of about 90 degrees in addition to excessive valgus of the femoral neck. The acetabulum was a shallow saucer filled with bone mixed with cartilage. It was not possible to bring the femoral head down to this level: so a socket was gouged out of the ilium immediately above the true acetabulum. The reconstructed

hip wa stable in abduction and medial rotation and was immobilised in plaster in that position. Intertr anteric adduction and lateral rotation osteotomy was carried out four months later and the plaste as finally removed after a further six weeks.

A ar later she was able to walk without pain and (with the shoe raised half an inch) without a consp ous limp. She had a stable hip joint with a negative Trendelenburg sign and less than an

FIG. 17

FIG. 18

Case 9. Figure 17—The hip three months after the infection began and before walking. Figure 18—At two years old after walking. The hip is dislocated and there is apparent loss of half the femoral neck, the epiphysial plate and the capital epiphysis.

FIG. 19

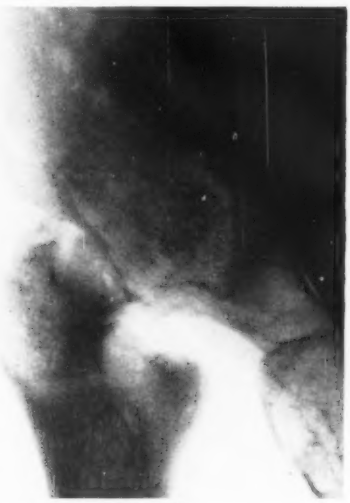


FIG. 20

Case 9. Figure 19—Arthrograph made at the same time as Figure 18. The surviving cartilaginous epiphysis is shown. The needle has entered the femoral head and the contrast so produced suggests that the epiphysial plate has survived as a translucent band distal to the opaque part of the head. Figure 20—One year after operation the now ossified head can be seen within the new acetabulum and the lower edge of a translucent line in the position of the epiphysial plate is visible.

inch f shortening well compensated by some residual fixed abduction. She had 20 degrees of fixed flex deformity and 20 degrees of flexion therefrom.

Co ent—Although this cannot be considered a wholly satisfactory outcome it compares fav rably with the alternatives of progressive shortening and instability. Furthermore, there no xists a situation which will favour successful arthrodesis should this become necessary in future (Fig. 20).

Case 10—A boy, aged thirteen months, presented one year after the onset of arthritis which h l been treated by antibiotics for one month at three weeks of age. An earlier radiograph (Fig. 21 at the time that the infection was subdued shows that the hip was dislocated and suggests that serious image

FIG. 21

Case 10—Radiograph taken soon after the onset of the infection showing that the hip is dislocated and serious damage seems to have been inflicted upon the upper end of the femur.

FIG. 22

Case 10—Condition one year later showing the extent to which the hip has improved radiologically.

had been inflicted upon the upper end of the femur. The dislocation was reduced by closed manip lation and one year later the promising situation shown in Figure 22 had developed.

Comment—This case demonstrates the success of closed manipulation as late as one yea after pathological dislocation had occurred.

Case 11—A girl, aged ten days, was seen three days after the onset and two days after streptococcal pus had been aspirated from the hip joint. The radiograph showed the upper end of the femur to be lying too high and too far laterally. A plaster was applied in 90 degrees of abduction and retained for three weeks during which the acute infection was controlled, though by now typical changes of osteomyelitis were visible above the acetabulum. She was seen at the Hospital for Sick Children when one year old with the femoral head still not visible on the affected side though well developed on the other. The ossific nucleus appeared at fourteen months and now (three years from the onset) closely resembles the normal. Shortening is trivial, the hip is stable, and movements are limited by one-third in abduction and rotation only.

Comment—This case illustrates the importance of early treatment in abduction and emphasises that absence of an ossific nucleus after it should have appeared does not mean that it is necessarily destroyed. Eyre-Brook (1960) drew attention to the importance of rest in abduction which, with aspiration and antibiotics, enabled him to obtain four good results in ten patients without the need for manipulation or operation later.

Case 12—A girl, aged two and a half years, was admitted to the Hospital for Sick Children having suffered acute arthritis due to staphylococcus aureus in infancy. Repeated aspiration yielded pus for one week after the beginning of antibiotic therapy. Three months later a sequestrum was removed

FIG. 23

FIG. 24

FIG. 25

Case 12—Radiograph one month after the onset of the infection. Figure 24—Appearance at the time of the operation. There is an apparent pseudarthrosis of the femoral neck but the capital epiphysis remains in the acetabulum. Figure 25—Appearance two years after exploration and abduction osteotomy. There is bony union at the femoral neck.

from the neck of the femur and this was followed by the drainage of pus for six weeks before final healing occurred. A radiograph (Fig. 23) shows the state of the upper end of the femur about a month after the infection was diagnosed.

On admission to the Hospital for Sick Children the radiographic appearances were as shown in Figure 24, when exploration was decided upon. At operation the femoral neck was found to be in fibrous continuity at the point of apparent pseudarthrosis; so an intertrochanteric abduction osteotomy was performed with medial displacement of the lower fragment. One year later sound union has occurred (Fig. 25) and the hip is stable. There is flexion through 80 degrees, full adduction and rotation, but no abduction from the neutral position. The leg is one inch shorter than its fellow.

Comment—Although the translucent area might well have recalcified if it had been left alone operation established again that translucency after osteomyelitis does not necessarily mean that bony tissue is lost, and the osteotomy both encouraged rapid remineralisation and reduced the varus deformity.

Case 13—An infant boy suffered an acute staphylococcal infection of the upper end of the femur which responded rapidly to antibiotics alone but later caused the hip to dislocate (Fig. 26). Closed manipulation was unsuccessful. At nine months he was admitted to the Hospital for Sick Children

FIG. 26

FIG. 27

Case 13. Figure 26—Radiograph showing the dislocated hip of a child aged ten weeks, six weeks after the onset of the infection. Figure 27—Condition at the time of operation at eight months of age. The capital epiphysis is ossified in the dislocated position.

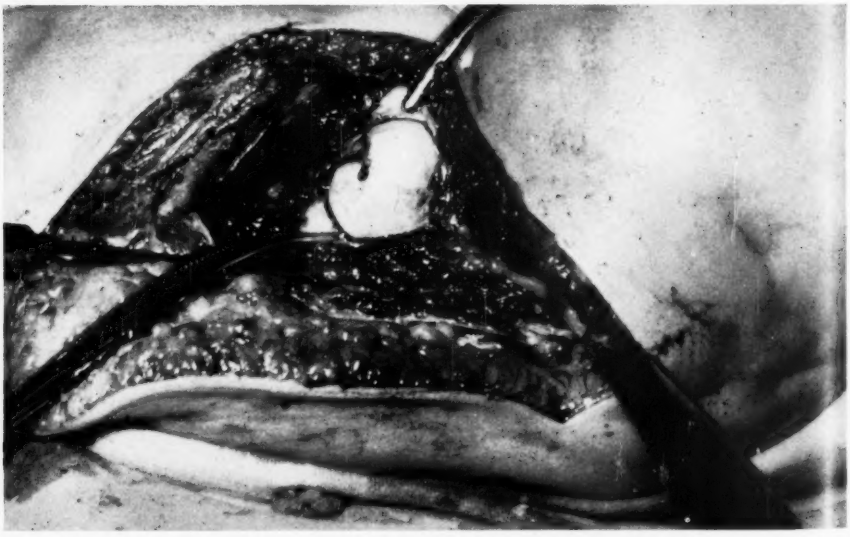


FIG. 28

Case 13—Photograph showing the head of the femur exposed at operation in the dislocated position. The forceps are gripping the capsule. The femoral head is relatively normal.

and th hip was explored. The femoral head was found to be dislocated but of nearly normal contour and c ered by apparently normal articular cartilage (Figs. 27 and 28). Reduction was achieved but the o ation was followed by recrudescence of the infection which has spoilt what promised to be a succe ul result. Nevertheless the femoral head has remained in the acetabulum and there is ankylosis in go position without shortening.

Com nt—Although this is disappointing the present state (Fig. 29) seems preferable to the alter ive of shortening and instability with the prospect of a difficult arthrodesis to be unde ken in the future. It is probable, however, that a longer interval should have been allow l between the acute infection and operation, for no such complication followed expl tion in Cases 9 and 12.

FIG. 29

Case 13—Condition one year after operation. Reduction and stability are maintained but recrudescence of the infection has further damaged the epiphysis and ankylosis is the result.

DISCUSSION ON AFFECTION OF THE HIP

It must be emphasised that neither dislocation nor destruction (apparent or real) of the hip joint necessarily contra-indicates an attempt to obtain a stable hip and reduce the shortening. At best the hip may be reduced by abduction alone or by manipulation; in less favourable cases open reduction into the original or a new acetabulum will decrease the ultimate disability. Osteotomy alone is clearly of benefit when the head survives in the acetabulum but part of the neck is lost. Eyre-Brook (1960) also illustrated its value when the head is lost but the neck remains in the acetabulum, and under certain other circumstances.

SUMMARY

Attention is drawn to the powers of resistance of the growing ends of bones in suppurative ogenic arthritis of the knee and hip in infancy.

It is emphasised that a translucent zone in the radiograph does not necessarily mean that s part of the bone, epiphysial cartilage or plate is destroyed.

Diagnostic criteria are described to confirm that cartilage or decalcified bone has survived infection in the knee joint.

The prognosis for the knee joint is discussed.

5. The hip joint presents greater difficulties in diagnosis and greater issues are at ake. It is recommended therefore that the hip joint be manipulated or explored if the radi raph and the physical signs suggest that destruction of the joint has either caused dislocation has so damaged it that dislocation is likely to occur in the future. In favourable cases st ility may be restored to the hip. In the others a diagnosis of irreparable destruction is estab hed and the surgeon is satisfied that an opportunity to help the patient has not been lost.

I am very grateful to the following for allowing me to report patients treated by them: Mr J. A. Cho eley (Case 3), Mr D. R. Urquhart (Case 10) and Mr R. H. Young (Case 7). Mr E. A. Devenish (Ca 11), Dr C. W. Kesson (Cases 1 and 9), Mr J. H. Meyer (Cases 5 and 6), Professor Alan Moncrieff (C 4), Dr J. Sakula (Case 12) and Mr D. R. Urquhart (Case 13) generously made available to me clinical de ls of the early course and treatment of patients who subsequently came under my care. Professor A. W. Wil son has kindly allowed me to follow the progress of the patient in Case 5, who is now under his care. It gi me pleasure to thank Mr Derek Martin for the clinical photographs and the reproductions of the radiogra s.

REFERENCES

BANKS, S. W., and COMPERE, E. L. (1941): Regeneration of Epiphyseal Cartilage. *Annals of Surgery*, **114**, 076.

BANKS, S. W., KRIGSTEN, W., and COMPERE, E. L. (1940): Regeneration of Epiphysial Centers of Ossific ion. *Journal of the American Medical Association*, **114**, 23.

EYRE-BROOK, A. L. (1960): Septic Arthritis of the Hip and Osteomyelitis of the Upper End of the Fem r in Infants. *Journal of Bone and Joint Surgery*, **42–B**, 11.

HAAS, S. L. (1919): The Changes Produced in Growing Bones After Injury to the Epiphyseal Cartilage Plate. *Journal of Orthopaedic Surgery*, **1**, 67.

HALL, R.M. (1954): Regeneration of the Lower Femoral Epiphysis. *Journal of Bone and Joint Surgery*, **36–B**, 116.

JONES, Sir R., and LOVETT, R. W. (1923): *Orthopedic Surgery*, p. 277. New York: William Wood & Company.

RECURRENT DISLOCATION OF THE PATELLA AND OF THE SHOULDER

Their Association with Familial Joint Laxity

CEDRIC CARTER, LONDON, ENGLAND

The Medical Research Council's Clinical Genetics Research Unit, Institute of Child Health, London

and

RODNEY SWEETNAM, LONDON, ENGLAND

Institute of Orthopaedics, Royal National Orthopaedic Hospital, London and Stanmore

Joint laxity is a feature of several dominantly inherited conditions. In Ehlers Danlos syndrome it is associated with hyperelasticity and fragility of the skin, in Marfan's syndrome with arachnodactyly, and in osteogenesis imperfecta with the well known bone changes. This laxity of ligaments may, however, occur alone. In any of these conditions the laxity of the joints may predispose to recurrent dislocation.

Joint laxity in more than one member of a family was first reported in the English literature by Finkelstein in 1916. The patient was a girl of two months with widespread generalised joint " hypermobility " and dislocation of both hips. Her mother, grandmother and two uncles also had joint laxity. A similar patient—a boy of seventeen months—was reported by Jahss in 1919, but the family history was less definite. The association of generalised joint laxity and recurrent dislocation of the shoulder has also been recorded (Sutro 1947), but there was no mention of any family inquiry.

In the most recent report on generalised joint laxity (Hass and Hass 1958) five patients were described, but none apparently had a relative similarly affected. Three of the patients had congenital dislocation of the hip, associated in one with dislocation of both radial heads and both inferior radio-ulnar joints. Of the remaining two patients, one had club feet and recurrent dislocation of the patella, and the other had dislocation of one sterno-clavicular joint.

In 1958 we drew attention to the association, in a single family, between familial joint laxity and recurrent dislocation of the patella (Carter and Sweetnam 1958). The pattern of inheritance of the joint laxity was that of a dominant gene.

That study led us to believe that joint laxity might be an important factor in recurrent dislocation of the patella and other joints, and that it might be found in a proportion of those patients with such dislocations. We now report a survey of patients with recurrent dislocation of the patella and with recurrent dislocation of the shoulder, designed to show how often these conditions are associated with, and are possibly caused by, familial joint laxity.

FAMILIAL JOINT LAXITY AND RECURRENT DISLOCATION OF THE PATELLA

We have traced the records of 111 patients suffering from recurrent dislocation of the patella who attended the Royal National Orthopaedic Hospital between 1948 and 1957. Letters were sent to each of them, asking whether any other member of their family had a similar condition.

Ninety-seven replies were received. Sixteen of the patients said that they had a relative similarly affected. All sixteen were examined, and a full family history was taken. In three instances two members of the same family replied, so that the total number of separate families was only thirteen. In three of these families we were unable to confirm the presence of recurrent dislocation affecting another relative, some other abnormality of the knee being discovered

instead. Thus, ten families remained, of which the member, or members, who had att ided the hospital for treatment constituted the index patients of this series. In two of the the underlying condition, both in the index patient and in the affected relative, was found be familial joint laxity.

As a check on the reliability of the answers to the questionnaire, and to examine me non-familial cases for joint laxity, home visits were paid to twenty of those who had ret ned replies saying that they had no relatives affected. On detailed family inquiry the answe hat no relative was affected was found to be right. Examination of these twenty patients sh wed that three had generalised joint laxity, and that in two of these the joint laxity, thoug not the dislocation, was present in other members of the family.

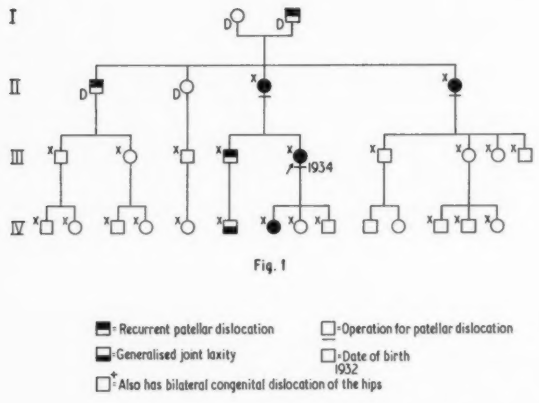


Fig. 1

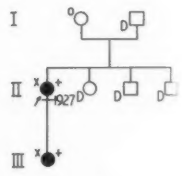
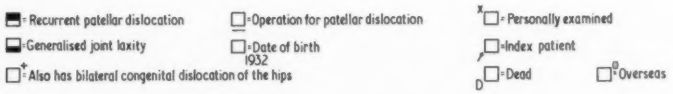


Fig. 2



FIGS. 1 AND 2

Pedigrees of the two families with recurrent dislocation of the patella associated with familial joint laxity.

Thus it appears that about 10 per cent of patients with recurrent dislocation of the patella have a relative similarly affected, and that familial joint laxity is not infrequently the cause of the recurrent dislocation when a relative is also affected. Familial joint laxity may also be the cause, though perhaps less often, of recurrent dislocation of the patella when there is no family history of dislocation.

In one other family with a pair of sisters who were both index patients the underlying condition was peroneal muscular atrophy (a condition known to be due to a specific dominant gene of varying manifestation).

In the remaining seven families, which contained more than one member with recurrent dislocation of the patella, but in which the index patient had no joint laxity, the affected relatives were as follows: son and daughter; aunt and mother; niece and uncle; mother; father; sister; niece.

The affected individuals in these seven families were carefully examined, clinically and radiologically. In some no abnormality could be discovered to which we could attribute the recurrent dislocation. In others only local abnormalities were found: for example, undue lateral mobility, underdevelopment of the patella, or hypoplasia of the lateral femoral condyle. No patient showed any significant knock-knee deformity. These local abnormalities were not always the same in the affected members of a single family, and their etiological significance is uncertain.

The two index patients and their families who had familial joint laxity are described below.

CASE REPORTS

Case 1—A woman aged twenty-four (III 5 in the pedigree shown in Figure 1) first dislocated her left patella at the age of six. There were many recurrences and two years later a similar dislocation started in the right knee. She was treated unsuccessfully at the age of fourteen by plication of the quadriceps expansion on the medial side of both knees. When she was sixteen the left patella was excised, and great capsular laxity was found at operation. At the same age she suffered a traumatic dislocation of the right elbow. The right patella still dislocates.

FIG. 3
Case 1—(III 5 in Figure 1). Laxity of the interphalangeal joints.

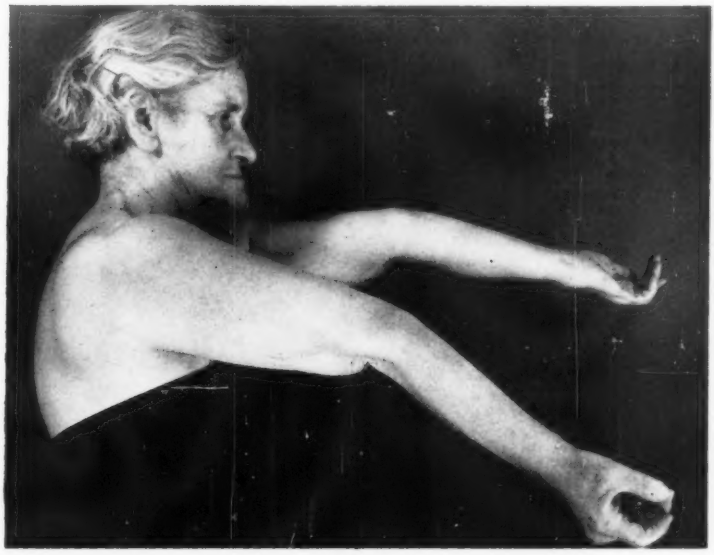


FIG. 4
Hyperextension of the elbows in the mother of the patient in Case 1 (II 3 in Figure 1).

On examination it was found that she had generalised joint laxity, the knees, elbows, wrists, and metacarpo-phalangeal joints all being involved (Fig. 3).

It is of interest that the patient's brother also had bilateral recurrent dislocation of the patellae, but had no generalised joint laxity. Such joint laxity was, however, present in his son. The laxity of the elbow joints in the mother (II 3 in Figure 1), a woman of fifty-five, is illustrated in Figure 4.

FIG. 5
Case 2—(II 1 in Figure 2). Joint laxity in the hand.

Case 2—A woman aged thirty-two (II 1 in the pedigree s own in Figure 2) was treated by Mr W. H. Trethowan for bil eral congenital dislocation of the hips at the age of two. hen seven years old she began to notice recurrent dislocati n of both knee caps. She has since suffered traumatic disloc ion of one shoulder, and has a permanent subluxation of oth radial heads. This patient presented a remarkable deg e of widespread joint laxity (Fig. 5).

Her only relative certainly affected was her daugh r (a girl aged twelve, III 1 in Figure 2), who also had bil eral congenital dislocation of the hips and recurrent sublux ion of both patellae. She, too, has generalised joint laxity the same degree as her mother.

The index patient's sister and her two brothers we all dead. Her mother was alive and well in New Zealan and she has lost touch with her father. None of these rel ives was considered by the patient to have had any joint disloc ion or joint laxity.

FAMILIAL JOINT LAXITY AND RECURRENT DISLOCATION OF THE SHOULDER

A similar inquiry was made of patients attending the Royal National Orthop dic Hospital, with recurrent dislocation of the shoulder, but these patients were lso asked if they, or any of their relatives, were double-jointed. Answers were received rom

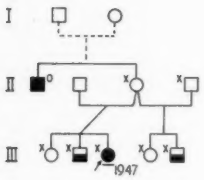


Fig. 6

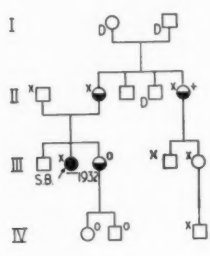


Fig. 7

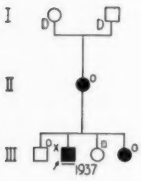


Fig. 8

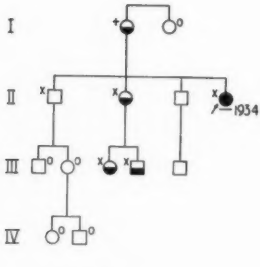
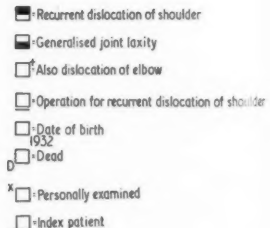


Fig. 9



FIGS. 6 TO 9
Pedigrees of the four families with recurrent dislocation of the shoulder associated with familial joint laxity.

forty patients, all of whom had attended for the first time between 1948 and 1957. Two of them reported that there was one relative similarly affected and also that other members of their respective families had double-joints. Another two reported that no relative was

similarly affected but that they had some relatives who were double-jointed. As a check on the reliability of the answers, home visits were paid to ten of those who stated that they had no relative affected, and the answers were confirmed. On examination of these ten patients, and on other patients with no family history, no generalised joint laxity was found. The case histories of the two patients with relatives similarly affected (Cases 3 and 5), and of the two patients with relatives who were double-jointed, but had no dislocations, are described below.

CASE REPORTS AND PEDIGREES OF INDEX PATIENTS

Case 3—A girl aged twelve (III 3 in the pedigree shown in Figure 6) dislocated her right shoulder when she was two years old. This recurred at about monthly intervals subsequently. Her left shoulder gave no trouble until eight years later, when it also dislocated. A successful Putti-Platt repair was performed on the right side in 1957.

Examination revealed generalised joint laxity. She had a somewhat lax skin and unusually short and broad feet. Unfortunately the mother and her brother were illegitimate, and so it was not possible to trace the family further back.

The patient's uncle aged thirty-one (II 1 in Figure 6), a seaman serving overseas, was not available for examination, but his medical records show that he has had treatment for recurrent dislocation of the right shoulder, and his younger sister gave a clear description of his generalised joint laxity.

FIG. 10
Case 3—(III 3 in Figure 6). Hyper-extension of the metacarpo-phalangeal joints.

Case 4—A woman aged twenty-seven (III 2 in the pedigree shown in Figure 7) knew from childhood that she and her sister were double-jointed. Her left shoulder first dislocated at the age of twenty, when she fell while skating. Subsequent dislocations occurred frequently, often without noticeable injury, and she was always able to effect reduction by herself. A Bankart operation was performed in 1957, and she has had no further trouble. She presented all the features of familial joint laxity.

The sister of the patient was in America with her children and was not available for examination, but the patient and her mother gave a convincing description of the sister's joint laxity.

Case 5—A man aged twenty-two (III 2 in the pedigree shown in Figure 8), who was a South African undergraduate at Cambridge University, first suffered dislocation of his left shoulder when twenty years old. Frequent dislocation, usually without even minor injury, occurred thereafter, often several times a month. A Bankart operation was performed six months after the first dislocation, and he has had no recurrences.

On examination he was found to have generalised joint laxity (one hand is illustrated in Figure 10). The family were not available for examination, but the patient reported that his mother and one sister were double-jointed, and that both had recurrent dislocation of the shoulder.

We have accepted this intelligent man's description of his affected relatives as sufficiently reliable for us to include the family in the series.

Case 6—A woman aged twenty-five (II 4 in the pedigree shown in Figure 9) first dislocated her right shoulder when she was about twelve years old. Frequent further dislocation followed and often occurred without warning, particularly when she was reaching forward with her right arm. When she attended hospital it was found that on bringing the elbow forward the right humeral head could be felt to slip backwards out of the joint (Fig. 11). Surgical repair of the capsule was performed at the age of eighteen; at operation the joint was exposed from the back and the labrum found to be loose. It was reattached to the roughened glenoid and the redundant capsule overlapped. Fourteen months later posterior dislocation recurred and a bone block was inserted. One year later, however, the humerus again dislocated, but, this time, forwards. A third operation was performed, and on this occasion the front of the glenoid labrum was found detached and the capsule extremely lax. An anterior repair was performed, and there has been no recurrence.

On examination she was found to have generalised joint laxity. Examination of other members of her family showed that, while none of them had had any joint dislocation, several had generalised joint laxity.

FIG. 11
Case 6—(II 4 in Figure 9). Posterior dislocation of the right shoulder.

DISCUSSION

Generalised joint laxity is not common. Two hundred orthopaedic out-patients were examined for the condition and only two were found to have generalised joint laxity of the degree found in the families reported in this paper. In one of the two it was a symptom of joint laxity, namely pain in the ankles from instability, which brought the patient to hospital.

It is clear from this investigation that joint laxity is a cause both of familial and sporadic cases of recurrent dislocation of the patella. There are, however, other ill-defined causes of the condition occurring in more than one member of a family. In contrast, familial joint laxity is rarely the cause of sporadic cases of recurrent dislocation of the shoulder, but, judging from our series, is the usual cause when the condition is familial.

The pattern of inheritance of generalised joint laxity in all these families is that given by a dominant gene or genes, as in those earlier reports in which the families were fully studied. There are some " skips " in the pedigrees in the sense that some of those who appear to have transmitted the gene are not themselves conspicuously loose-jointed; the reason may be that the diagnosis of joint laxity in mild degree is not easy. For example, the mother (II 3) of the index patient in Figure 6 with an affected brother has had affected children by two different husbands, but has no definite joint laxity herself. Again, the brother (III 4) of the index patient in Figure 1 has a son with generalised joint laxity, but he himself, though he has recurrent dislocation of both patellae, has only the laxity of the patellar ligaments that one associates with recurrent patellar dislocation.

There is no doubt that other joints may be affected in familial joint laxity. The mother and daughter shown in Figure 2 also had bilateral congenital dislocation of the hip, like the patients reported by Finkelstein (1916), Jahss (1919) and Hass and Hass (1958). The part played by familial joint laxity in cases of familial congenital dislocation of the hip is being further investigated by one of us. Congenital dislocation of the hip is not necessarily always present at birth, but the association of familial joint laxity with congenital club foot, reported

by Key (1927), Hass and Hass (1958), Carter and Sweetnam (1958) is interesting; for, unless the association of club foot and joint laxity in these patients is coincidental, it indicates that some joints are already abnormally lax before birth.

Failure to obtain satisfactory surgical repair in patients with recurrent dislocation of the patella or shoulder cannot often be due to generalised joint laxity. Dislocation recurred after operation in only one of the index patients in this series. This patient (II 4 in Figure 9) suffered from recurrent posterior dislocation of the shoulder, and recurrence followed fourteen months after the first operation. A second repair was followed by anterior dislocation, and this required yet another repair before stability was finally achieved. A similar recurrence in a patient with generalised joint laxity has been observed before by Osmond-Clarke (1959).

SUMMARY

An inquiry was made of ninety-seven patients with recurrent dislocation of the patella and forty patients with recurrent dislocation of the shoulder to see how often they had a relative similarly affected, and also how often such dislocation is associated with, and perhaps caused by, familial joint laxity.

Ten of those with recurrent dislocation of the patella and two of those with recurrent dislocation of the shoulder were found to have a near relative with a similar dislocation. Familial joint laxity was found in two of the ten families with more than one member affected by recurrent patellar dislocation, and in both those with more than one member with recurrent dislocation of the shoulder. Familial joint laxity was also found in two out of twenty patients with recurrent dislocation of the patella who had no family history of similar dislocation; but in none out of twenty patients with recurrent dislocation of the shoulder and who had no family history of similar dislocation.

Familial joint laxity may be the only cause of recurrent dislocation of the shoulder occurring in more than one member of the family. But there are other, as yet undefined, causes of familial recurrent dislocation of the patella.

We wish to thank the surgeons of the Royal National Orthopaedic Hospital for permission to investigate the families of their patients. It is with pleasure we acknowledge the help of Mr H. Jackson Burrows and Dr J. A. Fraser Roberts in the preparation of this paper. We should also like to thank the staffs of the Records Department and the Medical Photographic Department of the Institute of Orthopaedics, and the Department of Medical Illustration at the Hospital for Sick Children, for their help.

REFERENCES

CARTER, C., and SWEETNAM, R. (1958): Familial Joint Laxity and Recurrent Dislocation of the Patella. *Journal of Bone and Joint Surgery*, **40–B,** 664.

FINKELSTEIN, H. (1916): Joint Hypotonia. *New York Medical Journal*, **104,** 942.

HASS, J., and HASS, R. (1958): Arthrochalasis Multiplex Congenita. *Journal of Bone and Joint Surgery*, **40–A,** 663.

JAHSS, S. A. (1919): Joint Hypotonia. *New York Medical Journal*, **109,** 638.

KEY, J. A. (1927): Hypermobility of Joints as a Sex Linked Hereditary Characteristic. *Journal of the American Medical Association*, **88,** 1,710.

OSMOND-CLARKE, H. O. (1959): *Personal communication.*

SUTRO, C. J. (1947): Hypermobility of Bones due to " Overlengthened " Capsular and Ligamentous Tissues; a Cause for Intra-articular Effusions. *Surgery*, **21,** 67.

SIMULTANEOUS POSTERIOR DISLOCATION OF BOTH SHOULDER

Report of a Case

EDMUND SHEPHARD, MAIDSTONE, ENGLAND

Seven undoubted cases of simultaneous posterior dislocation of both shoulders have been reported (Coover 1932, Wilson and McKeever 1949, Dorgan 1955 (2 cases), Arden 56, O'Connor and Jacknow 1956, Goodfellow and Boldero 1959). Of these, five were caused by an epileptiform fit, and one by a road accident. In one, the cause was unknown. Manipulation was tried in five cases, but it was successful only in two. In both of these, treatment was undertaken at once (Dorgan 1955, Goodfellow and Boldero 1959).

A further case is here reported, in which manipulative reduction was possible after five days.

CASE REPORT

A muscular man aged fifty-six had been stooping while working with concrete in his garden. He felt unwell, came into the house and sat down. His wife then observed a fit, in which he

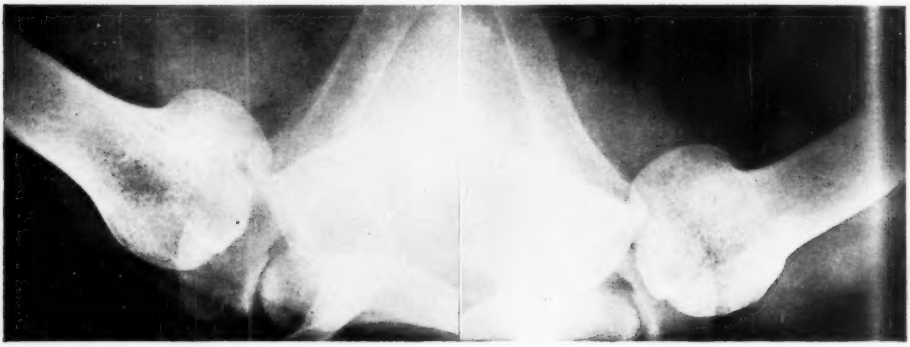


FIG. 1 FIG. 2

Vertical view of right (Fig. 1) and left shoulders showing bilateral posterior dislocation, the posterior glenoid rims engaging in notches in the humeral heads.

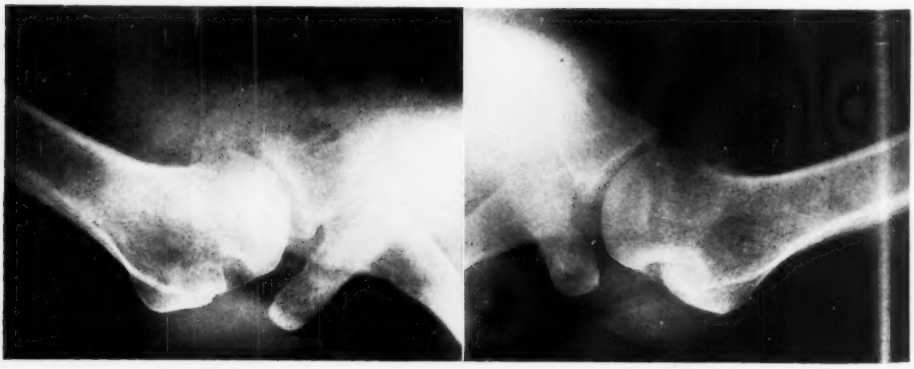


FIG. 3 FIG. 4

Vertical view after reduction shows that the indentations in the humeral heads correspond to the shape of the posterior glenoid rims, and it is concluded that these are impacted fractures. Figure 3—Right shoulder. Figure 4—Left shoulder.

kicked out his legs, flexed his elbows and contorted his face. Blood came from his mouth, and he was unconscious for five minutes. He did not pass urine during the attack.

On admission to the medical ward, he complained of severe pain in both shoulders. The left side of the tongue had been bitten, the left upper canine tooth was loose, and the gum was bleeding. There was a petechial rash over the shoulders and base of the neck. No abnormal neurological signs were found.

Four days after admission, orthopaedic advice was obtained. The arms were by the sides and the shoulders fixed in 60 degrees of medial rotation. The humeral heads were displaced backwards, leaving the acromion and coracoid processes prominent in front. An anteroposterior radiograph of the right shoulder, taken on the day after admission, suggested incongruity. Vertical views confirmed posterior dislocation of both shoulders, the posterior glenoid rims engaging in notches in the humeral heads (Figs. 1 and 2). Under anaesthesia, the dislocations were reduced easily, and collar and cuff slings were applied. Radiographs after reduction showed that the indentations in the humeral heads corresponded to the shape of the posterior glenoid rims (Figs. 3 and 4). It is concluded that these were impacted fractures; Goodfellow and Boldero so explained similar indentations in their case.

The shoulders were freed after four weeks. Twelve weeks after injury the shoulders were stable, and movement and function were full.

COMMENT

Simultaneous posterior dislocation of both shoulders is almost always due to an epileptiform fit. It is noteworthy that four patients, men of seventy years (Coover), forty-one years (Arden), forty-six years (Goodfellow and Boldero), and fifty-six years (the present case), had never had a fit before. Arden's patient and the present one had each been stooping at work in the garden just before the fit, no organic cause for which was found in our patient and none reported in the other three. Our patient has had no further fit in the subsequent eight months, nor were any more reported in the others. These unusual features suggest a possibility that the fits may be of a special nature. Their severity is indicated by the dislocation and also, in our case, by the petechial rash over shoulders and base of neck.

SUMMARY

A case of simultaneous posterior dislocation of both shoulders, occurring in an epileptiform fit, is described. Manipulative reduction succeeded after five days, and full function was gained.

REFERENCES

ARDEN, G. P. (1956): Posterior Dislocation of Both Shoulders. *Journal of Bone and Joint Surgery*, **38-B,** 558.
COOVER, C. (1932): Double Posterior Luxation of the Shoulder. *Pennsylvania Medical Journal*, **35,** 566.
DORGAN, J. A. (1955): Posterior Dislocation of the Shoulder. *American Journal of Surgery*, **89,** 890.
GOODFELLOW, J. W., and BOLDERO, J. L. (1959): Bilateral Synchronous Posterior Dislocation of the Shoulder. *British Journal of Surgery*, **46,** 413.
O'CONNOR, S. J., and JACKNOW, A. S. (1956): Posterior Dislocation of the Shoulder. *Archives of Surgery*, **72,** 479.
WILSON, J. C., and MCKEEVER, F. M. (1949): Traumatic Posterior (Retroglenoid) Dislocation of the Humerus. *Journal of Bone and Joint Surgery*, **31–A,** 160.

FLEXION CONTRACTURES OF THE KNEE

E. W. SOMERVILLE, OXFORD, ENGLAND

From the Nuffield Orthopaedic Centre, Oxford

Even such an apparently simple deformity as flexion contracture of the knee may present certain problems. These contractures can be divided into two types, and the method of treating one may not be effective in treating the other.

The first type is the simple contracture in which release of the tight structures by division or stretching allows the upper end of the tibia to glide round the femoral condyles into full extension.

In the second type, when the contracted structures are divided or stretched the upper end of the tibia hinges at its anterior edge instead of gliding round the femoral condyles. Thus the tibia becomes subluxated backwards on the femur (Figs. 1 to 3). This type of contracture is the more difficult to treat.

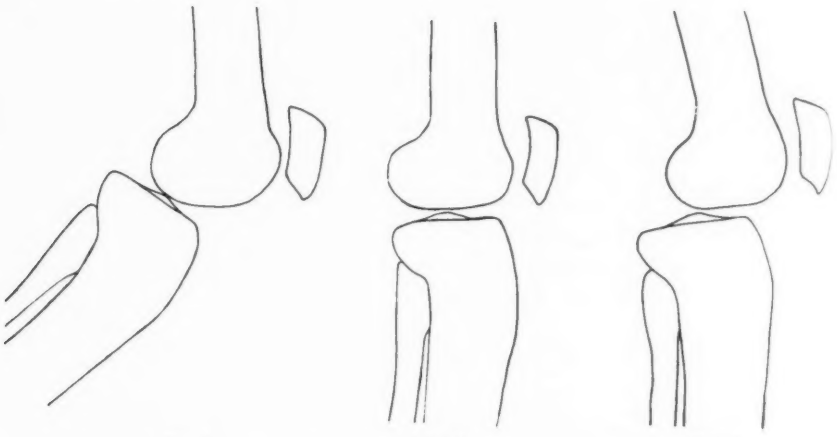


FIG. 1 FIG. 2 FIG. 3

Figure 1—The flexed knee. Figure 2—The knee has glided into extension. Figure 3—The knee has hinged into extension, with consequent backward subluxation of the tibia.

Sometimes it will be obvious from the start that backward displacement of the tibia is present but often it will become apparent only when the contractures have been overcome and extension can be attempted. To understand the reason for this it is necessary to review the functional anatomy of the knee.

The knee joint must serve two purposes. It must be capable of rigid stability in extension, but of free unrestricted flexion. To be able to provide these actions the lower end of the femur has two functionally different surfaces which may be termed the weight-bearing and the articulating areas.

The weight-bearing area is relatively flat, and when the knee is in full extension it fits exactly to the upper end of the tibia. Stability is provided by the collateral and anterior cruciate ligaments which are taut when the knee is extended, so that the two surfaces are firmly opposed. But whereas two flat surfaces firmly opposed by taut ligaments provide excellent stability the arrangement is unsatisfactory for free movement (Fig. 4).

he articulating area of the femur is almost exactly a half circle around which the tibial con les glide from full extension to full flexion. To allow this movement to be free the liga nts become relaxed as soon as flexion begins (Fig. 5). The relaxation occurs because the llateral and cruciate ligaments are attached eccentrically in the circle of which the articular

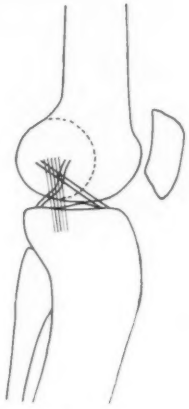


FIG. 4

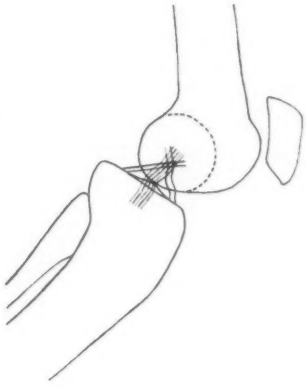


FIG. 5

Figure 4—The knee in extension. The collateral and anterior cruciate ligaments are taut. The weight-bearing surfaces are firmly opposed.
Figure 5—The knee in flexion. The collateral and anterior cruciate ligaments are relaxed. The tibial condyle is in contact with the articulating surface of the femoral condyle.

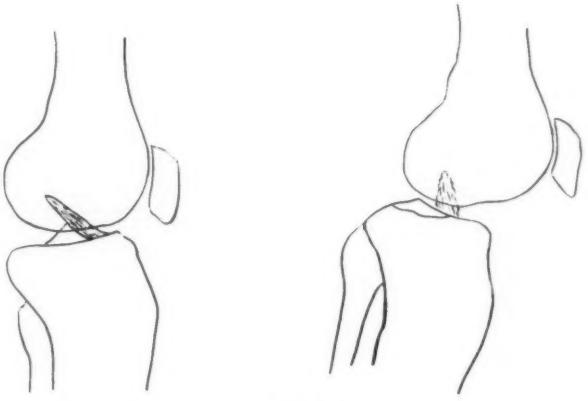


FIG. 6

The relaxed anterior cruciate ligament becomes permanently shortened. The tibia is displaced posteriorly on the femur.

a a forms over half. With this arrangement the anterior cruciate ligament is taut in extension a d lax in flexion, and the posterior cruciate is taut in flexion and lax in extension.

In cases of long-standing flexion contracture from injury or from an inflammatory c ndition such as rheumatoid arthritis the anterior cruciate ligament may undergo adaptive s ortening. This will prevent the last few degrees of extension by stopping the tibia from g ding forwards on the femur; so that hinging will occur instead (Fig. 6).

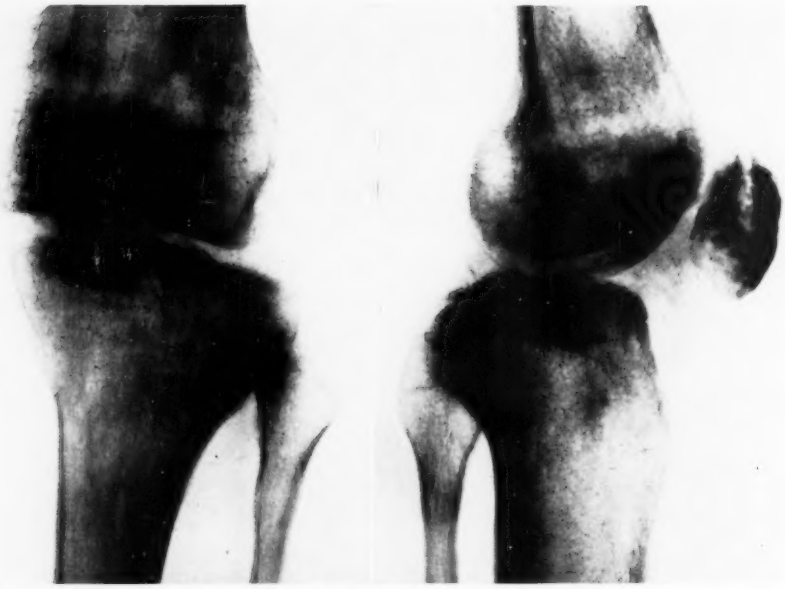


FIG. 7
Case 1—Traumatic subluxation of the knee. The tibia is displaced posteriorly and laterally.

FIG. 8
Case 1—After operation. The subluxation has been reduced.

TREATMENT

Unless gliding can be restored, normal full extension cannot be obtained. Several methods have been described for correcting the backward subluxation. These have usually been based on the principle of applying traction forwards at right angles to the upper end of the tibia at the same time as longitudinal traction. If the cruciate ligament can be stretched by these means the deformity will be corrected, but if it cannot be stretched the deformity can be corrected only by division of the ligament.

The operation is carried out with the knee flexed as for excision of a semilunar cartilage. The medial compartment of the knee is opened through a small anterior incision. The anterior cruciate ligament is picked up with a hook and divided. In some knees with rheumatoid arthritis the ligament may be difficult to find because it lies flat on the tibia and has become adherent to it; and it is difficult to get a hook round it.

After the ligament has been divided the tibia is pulled forcibly forwards on the femur with the knee flexed, as in testing the integrity of the ligament. Considerable force may be required to overcome all the other shortened structures. In most cases the after-treatment is the same as for the operation of excision of a semilunar cartilage, active movements being started as soon as the wound is healed.

RESULTS

This operation has been carried out twelve times; twice for persistent subluxation, the result of faulty splintage; twice for deformity after injury, and eight times for the flexion contracture from rheumatoid arthritis.

CASE REPORTS

Case 1—A man aged twenty-four years sustained an injury to his left knee when a crane fell across it. A diagnosis of rupture of the medial ligament was made. The knee was splinted in a plaster cylinder for three months, after which mobilisation was begun. After a month it was found that the knee was unstable, and it was clear that the medial ligament had become detached from the femoral condyle and was turned into the joint (Fig. 7). At operation the inturned ligament was with difficulty drawn out of the joint, but it was impossible to correct the backward and lateral subluxation of the tibia which was present. The cruciate ligaments had been ruptured and had united in a mass of scarred tissue. Only when this had been divided could the subluxation be reduced. Even then the reduction was unstable and the tibia kept displacing backwards. This was prevented by the introduction of a Steinmann's pin through the tibial tuberosity and its incorporation in a plaster cylinder applied from groin to ankle. After four weeks the plaster and pin were removed and active movements were started. Flexion well past 90 degrees was obtained. When seen eight years later the patient reported that he had been able to indulge in full athletic activities without any restriction (Fig. 8). The knee showed only slight instability.

Case 2—A woman of fifty stumbled and felt something " go " in her right knee, which she was subsequently unable to extend fully. During the next twelve months the knee was immobilised in plaster and manipulated on several occasions. At the end of this time the knee was flexed, painful and unstable (Fig. 9). An arthrograph showed that the medial semilunar cartilage was intact, but examination under anaesthetic showed that when the knee was forced into extension the tibia was displaced backwards on the femur and the displacement was more on the medial side than on the lateral. After the anterior cruciate ligament had been divided the tibia could be drawn forwards and when the knee was fully extended the reduction was stable. A plaster cylinder was maintained for three weeks. Mobilisation was rather slow but eventually almost full flexion was regained and very little instability remained (Fig. 10). Two years later the patient went on a mountain climbing holiday without trouble, although the knee is obviously arthritic.

FIG. 9
Case 2—The tibia is displaced posteriorly on the femur. The antero-posterior view shows that the displacement is chiefly on the medial side.

FIG. 10
Case 2—After operation. The posterior displacement has been corrected. The apparent loss of "joint space" on the medial side is much less marked.

Case 3—A woman of sixty-five sought advice for long-standing rheumatoid arthritis with such severe flexion contractures of the knees that she had not been able to walk for a year (Fig. 11). After a prolonged but unsuccessful attempt to correct the deformity by conservative methods both anterior cruciate ligaments were divided and the deformity was corrected without difficulty (Fig. 12). The left knee became completely stiff in good position. The right knee regained more than 90 degrees of flexion but in the course of four years 10 degrees of flexion contracture has recurred. She can walk unaided but usually uses one stick.

FIG. 11

FIG. 12

Case 3. Figure 11—Flexion contracture in rheumatoid arthritis. It could not be corrected by conservative methods. Figure 12—The same knee after division of the anterior cruciate ligament.

Of the other patients with rheumatoid arthritis, two (four knees) were successfully corrected immediately, in one (two knees) correction was obtained gradually after division of the anterior cruciate ligaments whereas previously no correction was possible, and in one (two knees) no correction was obtained. In this last patient the arthritis was in an active phase whereas in the others it was quiescent, which may have accounted for the failure.

SUMMARY AND CONCLUSIONS

1. The nature of flexion contractures of the knee joint is discussed.
2. It is suggested that division of the anterior cruciate ligament will help in the correction of the deformity when conservative methods have failed.
3. The results of the treatment of twelve such knees are mentioned and illustrative cases are described.
4. It must be emphasised that this method of treatment should be undertaken only when conservative methods have failed.

NEONATAL SCIATIC PALSY FROM INJECTION INTO THE UMBILICAL CORD

N. E. SHAW, LONDON, ENGLAND

From the Children's Hospital, Sheffield

Paralysis and gangrene of the lower limb in the newborn have been reported as occurring together or independently. Ehrenfest (1931) reported several of Bernhardt's cases of sciatic paralysis after extraction by the foot. He stated: "Bruns expressed the opinion that circumscribed paralyses of the lower extremity of newborn infants are probably not uncommon, but are overlooked or not reported, because they are likely to disappear promptly."

Michaëlis (1931) reported complete lateral popliteal paralysis of an infant of six weeks. The pregnancy and delivery had been normal, and the etiology was not discovered. Meyer (1931) described three children with neonatal paralysis of the lower extremity: one attributed to traction on the limb during breech delivery, one associated with fracture of the femoral shaft, and another complicating purulent meningitis that ultimately proved fatal.

Lateral popliteal paralysis in the right leg of a child delivered by Caesarean section was described by Lust and Berrewaerts (1932). The palsy recovered in twenty days and the cause was ascribed to "faulty intra-uterine position with compression." Mills (1949) described eight cases of neonatal sciatic palsy and referred to one other which he had traced. The etiology remained unproven, but it was suspected that the lesion might have been caused by injection of drugs into an umbilical artery. Bates and Page (1949) reported another child in whom nikethamide had been injected into the cord. Fahrni (1950) presented eleven examples of sciatic nerve paralysis; he suggested that direct intra-uterine pressure played a part in the etiology. Hudson, McCandless and O'Malley (1950) reported twenty cases including the eleven already reported by Fahrni.

The present paper reports three cases of neonatal sciatic palsy, two in a pair of twins and the third also in a twin. A review of the literature has failed to reveal another case in twins.

CASE REPORTS

Case 1—N. G. was admitted on July 5, 1957. He was the first-born of twin boys delivered by Caesarean section five hours before admission. Because of white asphyxia at birth, nikethamide (1 millilitre) had been injected into the umbilical cord. Soon afterwards a pinkish mottled discoloration developed in the skin of the left thigh and left iliac fossa. A left foot-drop was also observed.

On examination the weight was 5 lb. 10 oz. (2·55 kilograms), temperature 101 degrees Fahrenheit (38·3 degrees Centigrade), and pulse 166. The pinkish mottling was present on the skin of the left iliac fossa, the femoral triangle, the left popliteal fossa, and the left half of the scrotum (Fig. 1). The left foot was flaccid. The femoral pulse was palpable on both sides, but was weaker on the left. The left dorsalis pedis pulse was not palpable. The rest of the affected limb was warm and of the same colour as the right leg, and active movement at the left knee was present. A knee jerk was obtained, but the ankle jerk was equivocal. The cardiac and respiratory systems appeared normal.

Diagnosis—The signs were ascribed to spasm in the branches of the external iliac and femoral arteries from retrograde injection of nikethamide into the umbilical artery, with involvement of the parietal branch of the internal iliac artery to the sciatic nerve.

Treatment—Papaverine hydrochloride (5 milligrams) was given by mouth every six hours for twenty-four hours. Penicillin (250,000 units) was given intramuscularly six-hourly and streptomycin (150 milligrams) twice daily.

Progress—Three days after admission the baby vomited bile-stained fluid. Bright red blood and mucus were seen in a small amount of faeces. The left femoral pulse was impalpable. The discoloration was spreading to the left hypochondrium. Similar discoloration about one inch in diameter appeared in the skin of the right iliac fossa. This suggested a spreading thrombophlebitis. Intravenous fluid (4·3 per cent dextrose with 0·18 per cent sodium chloride) containing heparin (2,600 units–10 milligrams) was given at a rate of 275 millilitres daily. On the next day the baby developed ileus and his condition deteriorated. Intravenous fluid was continued and gastric aspiration commenced. Heparin was discontinued. After a further two days the infant was taking fluids by mouth and passing faeces. The skin over the left iliac

FIG. 1
The twins described in Cases 1 and 2. *Left*—N. G. (Case 1). *Right*—K. G. (Case 2).

fossa became hard and there was a suggestion of developing flexion contracture of the left hip and knee. Passive exercises and splinting were instituted. A week later he was fully breast fed and gaining weight (5 lb. 5 oz.=2·01 kilograms). He was discharged home.

On July 28, twenty-three days after his first admission, the infant was readmitted with a paracolic abscess pointing in the left iliac fossa. This was incised and drained. A faecal fistula developed, but the child's progress was satisfactory, and subsequently the fistula was closed. The foot-drop has persisted.

Case 2—K. G., the younger twin-brother of N. G. (Case 1) was admitted to the Children's Hospital on July 10, 1957. Although flaccidity of the left foot was reported on July 6 it was

two days later before he developed the pink cutaneous mottling like that seen in his brother. He had received an injection of nikethamide into the cord at birth.

On examination the birth weight was 5 lb. 11 oz. (2·8 kilograms), temperature 97·6 degrees Fahrenheit (36·4 degrees Centigrade), pulse 140, regular and the volume full.

Pink mottling was seen over small areas in the left iliac fossa and the upper quadrant of the femoral triangle, and over the medial aspect of the left knee and calf. All pulses were palpable. Foot-drop was present on the left. The skin mottling faded and no further signs developed.

Treatment—Passive movements of the left lower limb were carried out. Active movement of the limb returned in five days and the baby was discharged from hospital on July 8, eight days after his admission.

FIG. 2
Case 3.

Case 3—J. H. was admitted on January 5, 1957, three days after birth by Caesarean section after the normal delivery of a monster and the finding of another leg in the vagina. The child was suffering from white asphyxia at birth, and received an injection of nikethamide (1 millilitre) into the cord. Soon afterwards the left leg was white, and twenty-four hours later bruising was noticed in the mid-thigh (Fig. 2). On examination the left leg was cold and showed purplish mottling below the knee. The pulses of the limb were impalpable. The limb was flaccid. The haemoglobin was 6·5 grammes per cent.

Treatment—A blood transfusion of 52 millilitres was given. On January 18, 1957, left lumbar sympathectomy was performed, but the circulation did not improve. The limb became gangrenous and was amputated through the mid-thigh three days later. On February 25 the amputation stump was dry and clean. The haemoglobin content was 12·6 grammes per cent, red blood corpuscles 4·3 millions per cubic millimetre, white blood cells 17,500 per cubic millimetre, and sedimentation rate 44 millimetres in the first hour (Wintrobe). Platelets were 9,000 per cubic millimetre. A week later the child became pale and dyspnoeic, but responded to oxygen. Similar episodes occurred for a day or two, and the child died in an attack on March 8, 1957.

Necropsy—The cause of death was pulmonary oedema and inhalation of vomit. There was thrombosis of the left inferior epigastric artery which may have been related to the primary arterial defect in the left leg. No gross anatomical deformity was present.

DISCUSSION

These three cases illustrate the characteristics described in the literature.

Clinical picture—Most of the pregnancies described in the literature have been normal. There has been no common factor in foetal presentation or delivery, but in all cases there has been respiratory distress and the administration of an analeptic into the cord.

The condition may be seen immediately, but in two children six days elapsed before the palsy was noticed. The time of onset of the condition is difficult to assess because the mode

of presentation varies according to whether the first lesion to be observed is the cutaneous manifestation or the paralysis.

Cutaneous lesions—Discrete areas of purple mottled discoloration appear over the affected parts of the limb, buttock or abdominal wall. The area of mottling is outlined by a slightly deeper purple margin, which becomes indurated and may ulcerate. The remainder of the skin of the limb may be pale, cold, and anaesthetic in the sciatic distribution.

The skin lesion may resolve, or a variable area of skin may die and slough, leaving an ulcer which is slow to heal. In one of Fahrni's cases healing took two months.

Circulatory changes—Arterial pulsation may be absent from the pale, cold limb, but it nearly always returns later. Sometimes the arterial supply is so poor that gangrene occurs, as in the third case of the present series and in one of Mills's cases.

Peripheral nerve lesion—This was recorded in all but two of thirty-two cases. It was observed immediately after birth or several hours or days later. The right leg was involved in eleven cases and the left in twenty-one. The nerve involvement varies from a mild paresis to complete flaccidity, with loss of reflexes and sensation. The lateral popliteal nerve is commonly involved, with consequent foot-drop, and this was present in our series. The skin lesion does not invariably accompany even the most severe nerve lesion.

Intra-abdominal lesions—In one of the cases reported by Hudson, McCandless and O'Malley (1950) the inferior mesenteric vessels were thrombosed and the child died from peritonitis; necropsy revealed thrombosis of the right common iliac and inferior mesenteric vessels which had caused gangrene and perforation of the pelvic colon. This knowledge enabled us to expect thrombosis of the inferior mesenteric and internal iliac group of vessels supplying the pelvic colon, rectum and bladder. Watch was kept for blood-stained stools and ileus from peritonitis. This occurred in one of my cases, but the child survived in spite of the development of a paracolic abscess.

Prognosis—Neonatal sciatic palsy from injection into the umbilical cord is a serious condition. In this series only one child recovered completely, one has some residual paralysis requiring further orthopaedic care, and one died.

Of the patients reported by Mills (1949), one infant died from cerebral haemorrhage and one from peritonitis; one infant had residual paralysis of the anterior tibial muscles. The remaining five recovered completely.

In only three of the ten survivors in Fahrni's (1950) series was recovery complete. In the other seven limbs no useful recovery was obtained in the affected muscles in spite of many months of treatment.

Occasionally paralysis of the affected limb may recover promptly and completely, but a complete or partial sciatic paralysis often remains leaving disability resembling that from poliomyelitis with concomitant failure of limb growth. Hudson, McCandless and O'Malley (1950) pointed out that the most apparent and persistent feature in all the babies is foot-drop. There may be a gradual recovery during the first six to nine months, and in a few instances recovery is complete, but in most some foot-drop persists and growth of the foot and leg is retarded. At least half an inch of leg shortening and a small foot with impaired sensation may be expected. Some of these infants require orthopaedic care to correct equinus deformity and inequality of limb growth.

The severity of the abdominal complication depends on the extent of the thrombosis of the inferior mesenteric artery. One of Mills's patients died from peritonitis after necrosis and perforation of the pelvic colon, whereas in the patient reported here the child survived although a faecal fistula developed.

Pathology—McFarland (1950) commented that the common features in Fahrni's cases were fatal shock and the injection of an analeptic into the umbilical cord. He postulated that the injection of an analeptic into the umbilical artery instead of the vein could cause spasm or thrombosis of the hypogastric artery, and the internal and external iliac vessels and their

branches. In neonatal shock the umbilical vein is collapsed, empty and invisible, where s the two arteries are more readily discernible. The structures supplied by the first large bra h of the internal iliac artery include part of the sciatic nerve, and the muscles and the skin o the lower gluteal region. These are all involved in the syndrome.

The skin of the iliac fossa may be affected by spread of the injection into the ex nal iliac artery and the inferior and superficial epigastric vessels, and the scrotal or vulva kin through the superficial external pudendal trunks from femoral arterial involvement. The od supply to the pelvic colon may be jeopardised by arterial spasm or thrombosis of the in ior mesenteric artery because of retrograde spread of the injected fluid.

Mills suggested that the etiology depended on three factors: 1) the volume of fluid inj ed: 2) the vigour of the " milking " process, which determines how much of the fluid ente the foetal hypogastric arteries; 3) the infant's systolic blood pressure, which determines ho far the retrograde injection spreads before being swept peripherally.

There may be initial arterial spasm with subsequent thrombosis simulating that fter intra-arterial injection of anaesthetic solutions. Attempts have been made to reproduc the lesion in newborn kittens, and some produced a limp but the lesion itself was not demonstr ed.

In the cases reported here the drug used was nikethamide, as in the cases of Mills, nd Bates and Page. Cycliton was the suggested offender in the series of Hudson, McCandless nd O'Malley, who pointed out that the same effects have not been seen with lobeline.

Cohen (1948) reported that intra-arterial injections of thiopentone caused mottled bluish cutaneous patches, some of which merely blistered the skin whereas others sequestrated it, leaving sloughing ulcers. He suggested that the ill effects of intra-arterial thiopentone might be due to its alkalinity (p.H. 10·4–10·6). Cycliton (p.H. 4·92) and nikethamide (p.H. 5·77) are both acid in reaction. It is unlikely that acidity plays a significant part in these lesions, because no ill effects have followed the use of lobeline (p.H. 4·8) as an analeptic. Lobeline is an alkaloid whereas cycliton and nikethamide are synthetic. Nikethamide causes vasoconstriction by local action on the vessels, a property not found when Curtis and Wright (1926) investigated lobeline.

Both cycliton and nikethamide are used in 25 per cent solution. Although the compounds themselves may be harmless enough pharmacologically, solutions of such concentration might possibly irritate the intima and so cause arterial spasm and thrombosis. Intimal injury is the precipitating factor in thrombosis (Annotation, British Medical Journal 1950).

Nerves have a regional and a longitudinal blood supply; but, in the case of the sciatic nerve, Adams (1942) reported convincing evidence for the dominance of the regional over the longitudinal blood supply. In one infant in which the sciatic nerve was sectioned after death from enteritis, there were recent haemorrhages into the nerve fascicles, while distal to it the circumference of the fascicles was waxy, suggesting shrinkage.

Holmes, Highet and Seddon (1944) pointed out that there is no effective treatment for established ischaemic nerve degeneration. If the ischaemia is transient, causing Wallerian degeneration and only slight endoneural fibrosis, then spontaneous recovery may be expected. Temporary ischaemia, presumably due to vasospasm, would account for the gradual return of function in many of the babies.

Arterial spasm in the small sciatic artery followed by thrombosis would greatly reduce the blood supply to the sciatic nerve and the buttocks, with consequent paralysis and gangrene. **Treatment**—It appears from the evidence available that injection of the analeptic into the umbilical artery is the cause of neonatal sciatic palsy. This is preventable.

Arterial spasm may be overcome by antispasmodic drugs such as papaverine. The action of intra-arterial procaine is evanescent. Oral papaverine, alcohol and aspirin have been used in cases attributed to thiopentone with no dramatic improvement. In our case papaverine was of doubtful value, but it was only given orally.

Anticoagulant therapy is desirable. Heparin may be used to overcome early thrombosis. Intermittent administration of heparin is inadequate for threatened arterial thrombosis, and heparin is best given in a saline drip (Arterial Injuries, Medical Research Council 1944). Kisewetter and Shumacker (1948) confirmed that a constantly prolonged coagulation time is more desirable and better than a fluctuating one, particularly in relation to clotting in the smaller arteries.

In the prevention of equinus and scar contraction it is necessary to use carefully padded splints because of sensory loss. Dressings to ulcerated areas are also necessary. Orthopaedic supervision is needed at least until the end of growth, and stabilisation of the foot may be performed if necessary.

SUMMARY

1. Three new cases of neonatal sciatic palsy are reported in twins. They received identical treatment after birth; in the case of the first two for identical difficulties occurring after delivery. In twins there is a greater liability to neonatal shock and so a greater incidence of umbilical injections.

2. The clinical evidence and the necropsy findings in Case 3 support the hypothesis that intra-arterial thrombosis is caused by the injection of an analeptic into the umbilical artery.

3. The clinical picture is described, with a discussion on the pathology, treatment and the reason for recovery in some cases.

I am grateful to Dr C. C. Harvey and Mr R. B. Zachary for their help, encouragement and permission to publish these cases. I am also indebted to Mr H. Jackson Burrows for his helpful criticism of this paper.

REFERENCES

ADAMS, W. E. (1942): The Blood Supply of Nerves. *Journal of Anatomy*, **76,** 323.

ANNOTATION. NEONATAL ASPHYXIA (1953): *British Medical Journal*, **ii,** 328.

ANNOTATION. RESUSCITATION OF THE NEWBORN—A POSSIBLE DANGER (1950): *British Medical Journal*, **i,** 237.

ARTERIAL INJURIES (1944): *Medical Research Council War Memorandum* No. 13. London: His Majesty's Stationery Office.

BATES, C., and PAGE, A. P. M. (1949): A New Neonatal Syndrome. *British Medical Journal*, **ii,** 756.

COHEN, S. M. (1948): Accidental Intra-arterial Injection of Drugs. *Lancet*, **ii,** 361, 409.

CURTIS, F. R., and WRIGHT, S. (1926): Observations on the Action of Lobeline. *Lancet*, **ii,** 1,255.

EHRENFEST, H. (1931): *Birth Injuries of the Child.* Second edition. New York and London: D. Appleton & Company.

FAHRNI, W. H. (1950): Neonatal Sciatic Palsy. *Journal of Bone and Joint Surgery*, **32–B,** 42.

HOLMES, W., HIGHET, W. B., and SEDDON, H. J. (1944): Ischaemic Nerve Lesions Occurring in Volkmann's Contracture. *British Journal of Surgery*, **32,** 259.

HUDSON, F. P., MCCANDLESS, A., and O'MALLEY, A. G. (1950): Sciatic Paralysis in Newborn Infants. *British Medical Journal*, **i,** 223.

KISSEWETTER, W. B., and SHUMACKER, H. B., Jun. (1948): An Experimental Study of the Comparative Efficacy of Heparin and Dicumarol in the Prevention of Arterial and Venous Thrombosis. *Surgery, Gynecology and Obstetrics*, **86,** 687.

LUST, M., and BERREWAERTS, M. L. (1932): Un cas de paralysie obstétricale du membre inférieur droit chez un enfant né par césarienne. *Bruxelles-Médical*, **12,** 507.

MCFARLAND, B. (1950): Comment on Neonatal Sciatic Palsy (Fahrni). *Journal of Bone and Joint Surgery*, **32–B,** 47.

MEYER, M. (1931): Paralysie obstétricale des membres inférieurs. *Revue d'Orthopédie*, **18,** 767.

MICHAËLIS, L. (1931): Über Peroneuslahmung beim Neugeborenen. *Bruns' Beiträge zur klinischen Chirurgie*, **152,** 629.

MILLS, W. G. (1949): A New Neonatal Syndrome. *British Medical Journal*, **ii,** 464.

A CONGENITAL MALIGNANT SYNOVIAL TUMOUR OF BONE

R. WINSTON EVANS, G. E. THOMAS and NORAH M. WALKER, LIVERPOOL, ENGLAND

Certain malignant tumours such as the neuroblastoma, nephroblastoma, retinoblastoma and hepatoblastoma develop almost exclusively in young children. It is exceptional, however, for a sarcoma to arise during intra-uterine life and show evidence of an aggressive disposition even in the foetus.

In his review of the literature Wells (1940) accepted only twenty-nine reports of congenital malignant neoplasms as true instances of sarcoma observed at birth. Most of these (twenty-one cases) were sarcomas of the soft parts; only in six instances was there definite evidence that the tumour arose in bone or from periosteum. Of the others (two cases), although it was stated that the tumour involved an extremity, there was no clear evidence as to whether the lesion developed primarily in bone or in soft tissues.

The purpose of this communication is to describe a curious tumour of bone, difficult to label because of undifferentiated morphological features, yet presumed to be synovial in nature, involving almost the whole shaft of the right humerus and believed to have arisen at an early period during foetal life.

CLINICAL FEATURES

Immediately after uncomplicated birth it was noticed that the baby girl had a prominent swelling of the right arm. Apart from slight fluctuation posteriorly the mass was firm. The general condition of the infant was excellent and no other abnormality was observed.

Radiographs of the limb six hours after birth showed a pathological fracture caused by a destructive bone lesion of the middle third of the humeral diaphysis (Fig. 1). Subperiosteal bone spicules radiated outward in all directions but chiefly at right angles to the shaft of the bone. When the infant was six days old the fluctuant part of the growth was aspirated and a small amount of blood-stained fluid obtained.

The clinical and radiological findings indicated so strongly that the lesion was a malignant neoplasm, possibly an osteosarcoma, that amputation of the limb was advised, but the infant's parents refused consent to any surgical operation. As an alternative x-ray therapy was advised and the child received 700r given to each of two parallel fields (8×6 centimetres anteriorly and posteriorly) and 1,000r homogeneous dose through the arm. This had no effect and the swelling increased in size. Further radiographs showed progressive bone destruction and considerable increase in the size of the soft-tissue swelling (Figs. 2 and 3). The whole skeleton was radiographed as well as the lung fields but no other abnormality could be demonstrated. The infant's general condition remained satisfactory until the age of six months when she became fretful, lost her appetite and developed enteritis and pulmonary congestion. She died at the age of seven months.

The child's mother is still alive and well five years after the birth of this child.

NECROPSY FINDINGS

The right arm was greatly enlarged, its diameter exceeding the left by more than three times. The swelling consisted of a tumour, firm in consistence except posteriorly where it was cystic. The skin overlying the growth was on the point of breaking down into an ulcer.

Dissection showed that the humerus was replaced almost entirely by an encapsulated ovoid tumefaction measuring five inches in its long axis and two and a half inches in transverse

FIG. 1

Radiograph of right humerus six hours after birth. It shows a pathological fracture of the diaphysis with spicules of bone deposited at right angles to the shaft. There is also evidence of a soft-tissue swelling.

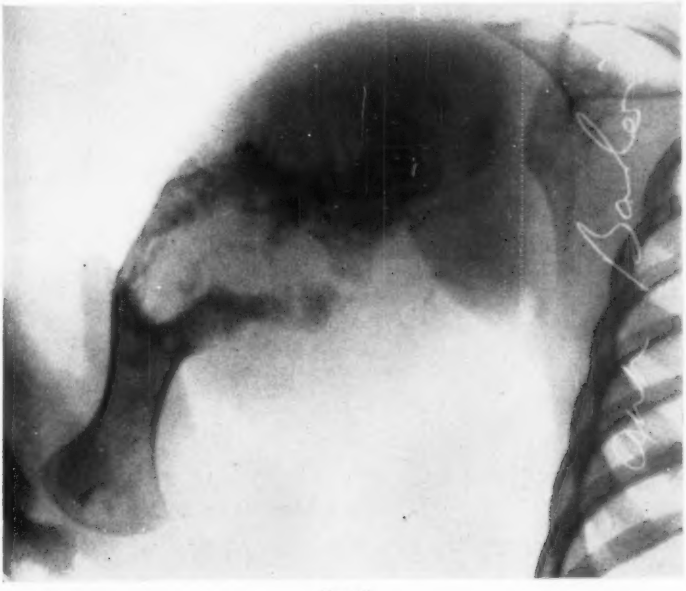


FIG. 2

Radiograph of right humerus five months after birth showing increased destruction of bone and a larger soft-tissue swelling. The proximal epiphysis has ossified normally.

and antero-posterior diameters. As judged by naked eye, only the upper and lower epiphysial cartilages were not invaded by neoplastic tissue. An extension of the neoplasm into the subacromial bursa had partly displaced the humeral head downwards into a dislocated position (Fig. 3). The peripheral margins of the tumour were limited, not everywhere distinctly, by expanded periosteum and compressed muscular tissue.

FIG. 3

Radiograph of right humerus seven months after birth, showing very marked destruction of the humeral diaphysis with remnants of bone in the soft tissues. The distal end of the humeral diaphysis remains but is slightly involved. The humerus is subluxated at the shoulder joint.

Neither the shoulder joint nor the elbow joint appeared to have been invaded by the growth. The axillary lymphatic nodes were enlarged but microscopic examination showed no more than non-specific reactive changes. No metastases were found either in lymph nodes or elsewhere in the body. The sympathetic ganglia and the adrenal gland showed no evidence of tumour. All other organs were normal in appearance except the lungs, and death was attributed to extensive bronchopneumonia.

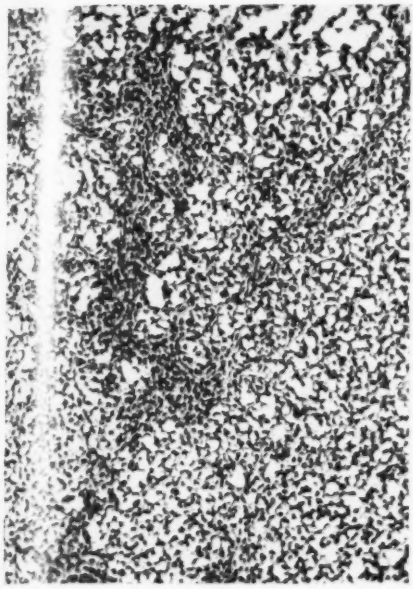


FIG. 4

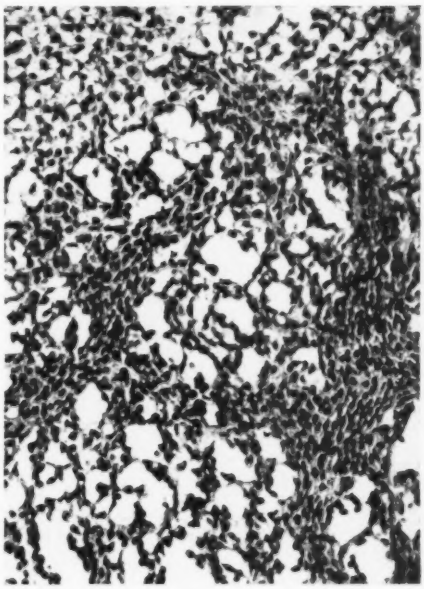


FIG. 5

Figure 4—General microcystic structure of tumour showing an intimate mixture of small "synovial" cavities and spindle cells arranged in bands. (Haematoxylin and eosin, ×116.) Figure 5—Detail of Figure 4 showing relationship of synovial cavities to fasciculated spindle cells. (Haematoxylin and eosin, ×180.)

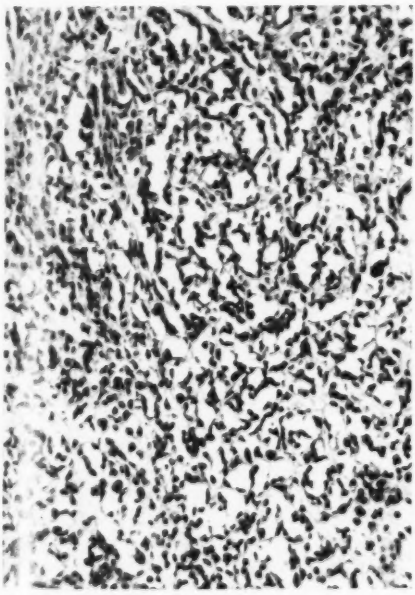


FIG. 6

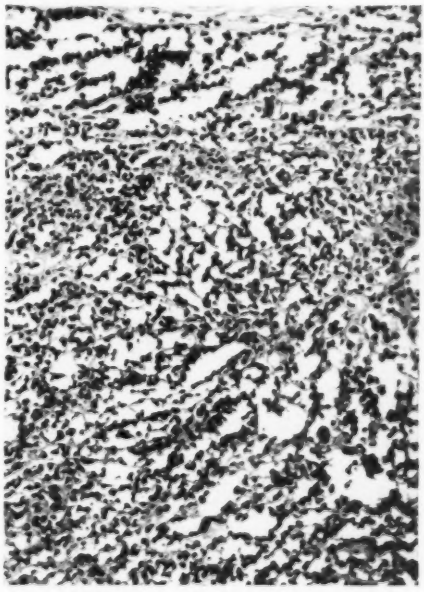


FIG. 7

Figure 6—General "angiomatoid" pattern. (Haematoxylin and eosin, ×145.) Figure 7—General structure with nuclei of cells lining cavities projecting as hobnails into the spaces. (Haematoxylin and eosin, ×110.)

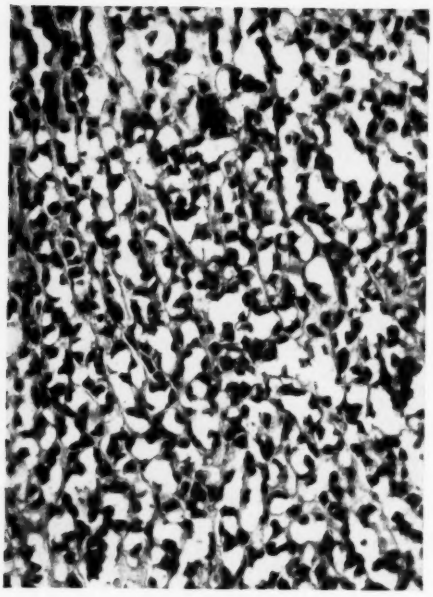


FIG. 8

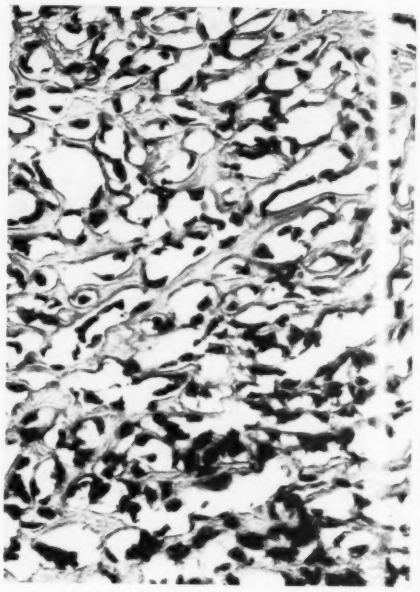


FIG. 9

Figure 8—Cavities, often ill-defined, separated by thin collagenous stroma. (Haematoxylin and eosin, ×175.)
Figure 9—Cavities separated by osteoid tissue. (Haematoxylin and eosin, ×220.)

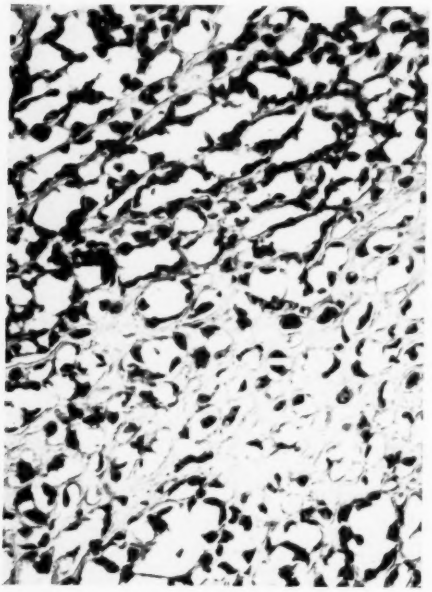


FIG. 10

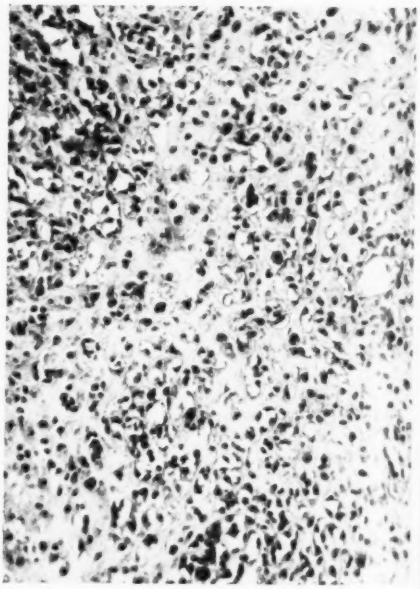


FIG. 11

Figure 10—Synovial cells encapsulated by cartilaginous matrix. (Haematoxylin and eosin, ×260.) Figure 11—Cells and cavities embedded within a chondromucinous substance. (Haematoxylin and eosin, ×17[illegible])

THE TUMOUR

The large ovoid tumour, firm in consistence, showed on its cut surface an opaque, grey white, fleshy tissue intermingled with areas of maroon colouring. Both bone marrow and bone cortex were replaced and destroyed by the neoplastic tissue which here and there had penetrated through the expanded periosteum. Despite this marginal infiltration the outline of the tumour was sharply defined and smooth. Tiny spicules of bone extended outside the shaft of the humerus within the neoplastic mass.

The histological features were chiefly of undifferentiated character and the general appearance was that of a vacuolated and microcystic tissue broken by small bands of spindle cells (Figs. 4 and 5). The microcystic holes lay close together; they were not sudanophilic but rather resembled endothelial channels and cavities lined by several cells with nuclei projecting as hobnails into the tiny empty spaces (Figs. 5 to 7). These microcysts, though varying in size, were nowhere very large. Mostly they were separated by a sparse connective tissue (Fig. 8) and delineated clearly by reticulin. In some areas there was pale and dense tissue of an osteoid, occasionally osseous, character interspersed between them (Fig. 9). In other areas the tiny channels fused into a chondromucinous matrix within which one or more luminal cells were enclosed either within poorly defined lacunae or by well defined capsules surrounded by a more mature type of cartilage (Figs. 10 and 11). The bone spicules and strands appeared inert, with no osteoblasts or osteoclasts in association with them, suggesting absence of osteogenesis and osteolysis.

Where the endothelial-like cavities bordered on groups of spindle cells arranged in bands the impression gained was that the cells lining the channels and spaces were arising from such connective tissue cells (Fig. 5). Occasionally, instead of forming spaces, stellate cells emerged and anastomosed by delicate protoplasmic filaments. No mucinocarminophilic substance was present within the lined spaces and the tumour tissue was periodic-acid-Schiff-negative.

A section taken at the upper extremity of the lesion and including part of the proximal epiphysial cartilage showed that the neoplastic tissue in this region was limited by tendons, and immediately adjacent to them were large nerves, some of which were infiltrated by tumour cells. Around the greatest transverse diameter of the growth the periosteum was penetrated and the tumour tissue spread for a short distance into compressed striated muscle.

DISCUSSION

The growth was a malignant mesenchymal tumour which, though in the main poorly differentiated, was substantially synovial in nature with a tendency to develop angiomatoid rather than membranous features (Fig. 6). However, it is not against expectation that synovial tissue in its deviated neoplastic form should produce angiomatoid patterns rather than membranous elements lined by epithelial-like cells. Other mesothelia such as the pericardium and peritoneum also are capable of giving rise to growths with angioformative properties.

There were areas of chondrosarcomatous differentiation in the tumour and these reflect the capacity, sometimes observed in synovial neoplasms in adults, for metaplastic change to cartilage and even bone (Evans 1956).

Although malignant synovial tumours developing in relation to joints tend to invade bone, and though in this case the tumour tissue abutted the tendons particularly in the region of the upper epiphysial plate, the general symmetrical shape, size and centrifugal manner of expansion of the growth, as well as the initial radiographic localisation in the middle third of the humeral diaphysis, suggest strongly that it arose endosteally as a primary tumour of bone. Yet it is admitted that one cannot state with certainty how this neoplasm arose and, though not considered probable, there is still a possibility that the growth may have invaded bone from an initial juxta-osseous tenosynovial origin.

In the embryo synovial tissue is formed by mucinous differentiation of a mesenchymal mass bounded on each side by the cartilaginous primordia of bone, themselves initially

developed from the same mesenchymal block. Therefore it is not surprising that a close embryological and anatomical relationship of these two tissues, synovial and osseous may be recapitulated in neoplasms developing from them. Nevertheless, despite the great morphological variations exhibited by bone tumours, evidence of synovial differentiation is seldom observed in them.

Unlike the two cases of synovial sarcoma of the tibia described by Hicks (1954), and the case of malignant synovioma invading the tibia reported by Lederer and Sinclair (1954), no histological resemblance to an adamantinoma-like pattern was observed in this tumour.

Synovial sarcoma is an uncommon tumour and it is especially rare in young children. The youngest patient in the series of ten cases of malignant synovioma described by Crocker and Stout (1959) was aged two years. Tillotson, McDonald and Janes (1951) reported a synovial sarcoma of the foot in a child one month old. The tumour was present at birth and was cured by local excision, the patient living without recurrence for twenty years. Adair (1935) referred to a malignant synovioma of the right knee in a child aged nine months. The tumour was first noticed when the boy was only two months old; it recurred after excision but after a second local resection the child was still alive and well three years later. Pack and Anglem (1939) also described a malignant synovioma in a child aged less than one year; like Adair, these authors gave no details of the histological features of the tumour. Wright (1952) described a poorly differentiated synovial sarcoma of the buttock in a child aged one year who died eight months after operation.

Of the forty-three cases of juvenile synovial sarcoma discussed by Crocker and Stout (eleven personal cases with thirty-three collected from the literature) there were sixteen instances in which the upper extremity was involved, but no example involving the upper arm. These authors chose cases only of the usual well differentiated type of malignant synovioma with pseudo-epithelial lined spaces and ramifying clefts intimately blended with a sarcomatous spindle-cell tissue. Histologically such tumours pose no diagnostic problems because their structure is characteristic, but it is to be expected that a tumour of bone exhibiting synovial differentiation and arising in the foetus would be predominantly primitive in structure with synovial characters not well developed.

SUMMARY

A case of synovial sarcoma of the humerus is described. The histological features were of an undifferentiated character and the tumour arose in the foetus.

REFERENCES

ADAIR, F. E. (1935): Tumor of Synovial Membrane of Knee-joint. *Annals of Surgery*, **101**, 811.

CROCKER, D. W., and STOUT, A. P. (1959): Synovial Sarcoma in Children. *Cancer*, **12**, 1,123.

EVANS, R. Winston (1956): *Histological Appearances of Tumours*, p. 90. Edinburgh and London: E. & S. Livingstone Ltd.

HICKS, J. D. (1954): Synovial Sarcoma of the Tibia. *Journal of Pathology and Bacteriology*, **67**, 151.

LEDERER, H., and SINCLAIR, A. J. (1954): Malignant Synovioma Simulating Adamantinoma of the Tibia. *Journal of Pathology and Bacteriology*, **67**, 163.

PACK, G. T., and ANGLEM, T. J. (1939): Tumors of the Soft Somatic Tissues in Infancy and Childhood. *Journal of Pediatrics*, **15**, 372.

TILLOTSON, J. F., MCDONALD, J. R., and JANES, J. M. (1951): Synovial Sarcoma. *Journal of Bone and Joint Surgery*, **33-A**, 459.

WELLS, H. G. (1940): Occurrence and Significance of Congenital Malignant Neoplasms. *Archives of Pathology*, **30**, 535.

WRIGHT, C. J. E. (1952): Malignant Synovioma. *Journal of Pathology and Bacteriology*, **64**, 585.

THE NATURAL HISTORY OF HALLUX VALGUS IN ADOLESCENCE AND EARLY ADULT LIFE

HARRY PIGGOTT, LONDON, ENGLAND

From the Royal National Orthopaedic Hospital and the Institute of Orthopaedics, London and Stanmore

In ordinary orthopaedic practice hallux valgus is seen much more frequently in adults than in adolescents. Yet when 100 consecutive unselected patients aged between thirty and sixty-five years were asked the simple question, " When did you first notice that your big toe was starting to point outwards? " fifty-seven said in the 'teens or earlier, thirty-eight did not know and only five said after the age of twenty. Hardy and Clapham (1951) similarly found that 66 per cent of adult patients remembered deformity before they were twenty years old. When variations in the accuracy of patients' observation and memory are allowed for, it therefore seems clear that the deformity commonly (perhaps usually) starts in adolescence or childhood, though many patients do not seek surgical help until symptoms become troublesome years later. In a community which shows an ever-increasing interest in its health it is likely that the proportion of patients seen early will increase.

Symptoms in early life are usually not severe and treatment therefore is mainly prophylactic, attempting to prevent progressive deformity and pain. It follows that we cannot logically select patients for operation unless we can forecast which are likely to develop such progression. The necessity for good selection is emphasised by the fact that attempts at operative correction are not always successful (Bonney and Macnab 1952).

A study has therefore been made of patients with hallux valgus who had first been seen during the growing period and who had not at any time had surgical treatment. An attempt has been made to trace the natural evolution of the deformity, with particular attention to any feature which in the early stages might serve as a guide to the structural prognosis of the individual foot.

It is admitted that the severity of symptoms that may develop with the march of time often does not parallel the degree of structural change: variations in personality, mode of life and subjection to fashion all play their part, so that one patient with very mild hallux valgus may be full of complaints whereas another with grossly disorganised forefeet has had perfectly good and comfortable service from them all her life. But, on the whole, patients with severe hallux valgus and associated changes in the forefoot such as increased width, depression of the anterior arch and dorsal subluxation of the lesser toes have more trouble than those with only mild valgus of the great toe and with the rest of the forefoot practically normal; certainly they present a more difficult therapeutic problem. This approximate correlation between deformity and symptoms when we consider groups of patients rather than individuals has been borne out by this study, and it is believed that knowledge of the structural prognosis of the individual foot when seen in adolescence should be of considerable help in determining the early management of these cases.

MATERIAL

All patients who had been seen at the Royal National Orthopaedic Hospital because of hallux valgus before they were twenty-one years old, and of whom adequate records were still available, were asked to attend for examination if their feet had never been operated upon. The upper age limit of twenty-one was selected because at that age all the epiphyses in the foot are closed.

It was necessary also to draw a somewhat arbitrary distinction between the slight valgus deviation of the normal great toe and hallux valgus in its usual sense. Hardy and Clapham

(1951, 1952) in extensive radiological studies on presumed normal subjects found that the mean valgus deviation of the great toe was 14·4 degrees at the age of fourteen to fifteen years, and 15·7 degrees in young adults; thus for this study a valgus of less than 15 degrees, measured radiologically, has been considered normal. This matter of distinguishing between normal and pathological valgus will be referred to again later when a natural differentiation based on anatomical grounds will be proposed, but in the meantime it may be pointed out that the artificial dividing line of 15 degrees accords remarkably well with clinical practice. Of the 116 patients examined only three were excluded for this reason; they had been recorded originally by different observers as having mild, minimal and ? hallux valgus. Of the remaining 113 patients ten had only one foot affected; so there were 216 feet for study.

The youngest patient seen was seven years old, and the oldest attending for subsequent examination was thirty-six; the longest period of observation in the individual was twenty-one and a half years, with an average of five years and one month. There were 110 women and three men.

All feet were radiographed at the final examination by the standardised technique described below. At previous routine out-patient attendances some had been radiographed in the same way, some by normal unstandardised methods, and some not at all. The radiological material available was therefore: 216 feet with final standardised radiographs; seventy-six feet with one or more earlier standardised radiographs taken up to six years before; and thirty-four feet with non-standardised radiographs up to fourteen years before the time of follow-up. When radiological measurements are referred to in this paper they have been taken from standardised films only.

METHOD

In the taking of the history the patients were asked at what age the deformity was first noticed, the nature and severity of the symptoms, the type of footwear, occupation, conservative treatment, and whether there was a family history of hallux valgus. The whole of both lower limbs were examined, particular note being taken of the degree of hallux valgus, rotation of the great toe around its longitudinal axis, movements of the first metatarso-phalangeal joint, medial projection of the head of the first metatarsal and other deformities or postural abnormalities such as hammer toe, pes planus anterior, pes valgus, and tibial rotation.

Antero-posterior radiographs were taken with both feet together on a horizontal cassette, the ankles being in 20 degrees of plantar flexion; the tube-film distance was forty-eight inches, the centre ray passing vertically between the first metatarsal heads. The long axes of the first and second metatarsals were marked on these films by bisecting the diameters of the proximal and distal ends of their shafts and joining the points thus obtained. The shaft of the proximal phalanx of the great toe is too short and irregular for this method to be applied: so its long axis was marked by joining the midpoints of its proximal and distal articular surfaces. It was then possible to measure the angle between the axes of the first metatarsal and the proximal phalanx (hallux valgus), that between the first and second metatarsals (intermetatarsal angle), and the inclination of the articular surfaces of the first metatarso-phalangeal joint to the long axes of their supporting bones (Fig. 1). Murray (quoted by Ellis 1951) considered that such angular measurements were accurate to within 1 degree, and repeated marking and measuring of the same angles as a preliminary to the present study confirmed this. Even so, it is not considered justifiable to base conclusions on comparisons of individual radiographs, even though the measurements themselves are accurate, because such factors as minor degrees of forefoot rotation, which vary from foot to foot, may affect the radiographic appearances, and it is at least conceivable that other factors, such as fatigue, may produce real changes in some of the angles being measured. However, if we compare large groups of cases as opposed to individuals these sources of variation are common to all, and differences between groups may be considered significant.

Among the other radiological features studied the most important was the presence or absence of subluxation at the first metatarso-phalangeal joint; many other factors, including

the degree of sesamoid displacement, were recorded, but only those which appear to be significant will be discussed.

RESULTS

Different types of valgus deformity—There were three distinct patterns in the relationship of the articular surfaces of the first metatarso-phalangeal joint to each other as seen radiologically. In the first, these surfaces were completely congruous as in the normal foot, their central points lying opposite each other. In the second the distal articular surface was deviated laterally on the proximal articular surface, leaving the medial end of the latter exposed. In the third the base of the proximal phalanx was subluxated laterally off the metatarsal head (Figs. 2 to 4).

FIG. 1 FIG. 2 FIG. 3 FIG. 4

Figure 1—Illustration of the method of marking radiographs for angular measurements (see text). Figures 2 to 4—Examples of the three different patterns of relationship of the articular surfaces of the first metatarso-phalangeal joint. In Figure 2 they are completely congruous; in Figure 3 the phalangeal surface is deviated laterally on the metatarsal head, leaving the medial end of the latter uncovered; in Figure 4 there is subluxation.

These three groups will be referred to as the congruous, deviated and subluxated groups respectively. In the earlier stages of this investigation it was thought that the congruous and deviated patterns (Figs. 2 and 3) might be produced by different postures of the same joint, but repeated radiographs of any particular foot (some were radiographed as many as five times) never showed a change from one type to the other; moreover it will be recalled that in spite of the existence of the so-called abductor and adductor muscles of the great toe, active side-to-side movement does not normally occur at the first metatarso-phalangeal joint.

As judged from the earliest available antero-posterior radiographs there were twenty feet in the first group, eighty-one in the second and 114 in the third. It will be observed therefore that well over half of these feet showed subluxation when first seen, in a few patients even before the age of ten years. In one foot, the head of the first metatarsal being of very irregular shape, it was impossible to define the limits of its articular surface and thus to allocate it to any group.

It might be thought at first sight that these three patterns represent different stages of the same condition, and indeed the evidence to be presented leaves no doubt that the deviated type may change into the subluxated; the congruous type, on the other hand, shows fundamental differences from the other two, and is best regarded as a separate entity. The vital distinction lies in the site at which the valgus deformity occurs. In the normal foot the articular surfaces of the first metatarso-phalangeal joint are not usually set strictly at right angles to the long axes of their supporting bones; if the general lines of the articular surfaces are marked by joining their medial and lateral ends we find that that of the metatarsal head is tilted slightly laterally on the shaft, and the shaft of the proximal phalanx is again tilted slightly laterally on its own articular surface (Fig. 5). The summation of these two small lateral tilts (angles A and B in Figure 5) accounts for the slight valgus position of the normal great toe, the joint surfaces themselves being congruous and parallel. Increased valgus can be produced either by greater tilting of one or both of the articular surfaces in relation to their supporting bones or, quite differently, by displacement at the joint itself, the phalanx moving laterally round (or off) the head of the metatarsal (Figs. 6 to 8). It will be seen that in the patients with congruous joints (Fig. 6) the whole of the valgus is accounted for by tilting of the articular surfaces—that is, by variations in the shape of the *bones* (either proximal phalanx, metatarsal, or both), whereas in the other two groups (Figs. 7 and 8) the deformity occurs at the *joint*. Thus the congruous type shows only exaggeration of a normal feature, whereas the deviated and subluxated types show different degrees of an undoubted abnormality – displacement of the articular surfaces on each other.

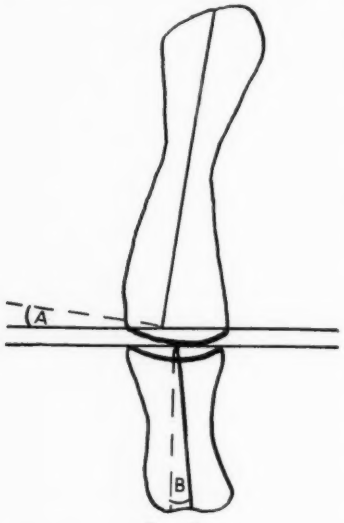


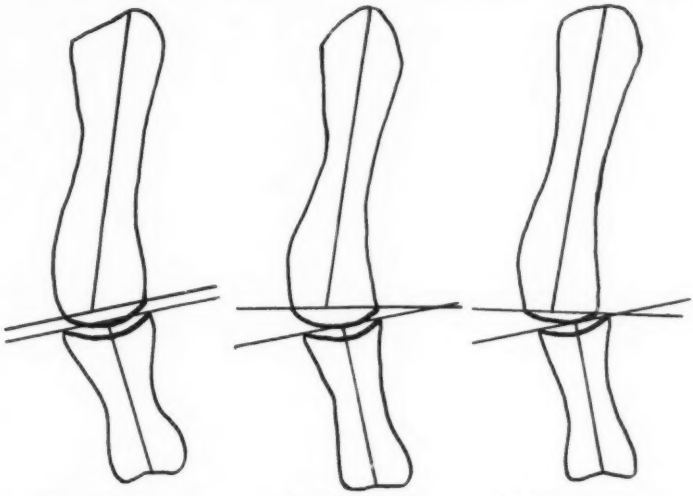
FIG. 5

Tracing of a radiograph of a normal foot, showing how the slight valgus alignment of the proximal phalanx is accounted for by the articular surfaces of the first metatarso-phalangeal joint being not quite perpendicular to the long axes of their supporting bones. The degree of hallux valgus is the sum of angles A and B.

It is possible of course that some of the deviated and subluxated toes could have excessive tilting of the articular surfaces as well as displacement at the joint, but the following considerations make this unlikely. If the angles A and B shown in Figure 5 are added together the sum will represent the amount of valgus due to tilting of the joint surfaces—that is, to variations in bone contour as opposed to displacement of the phalanx; this applies to all types, whatever the relationship of the articular surfaces to each other may be. Figure 9 shows that this element of the deformity in the deviated and subluxated toes varies from 0 to 34 degrees with a mode of 15 degrees, whereas in the congruous group (where it accounts for the total valgus) it covers only the upper half of this range. The distribution chart for the deviated and subluxated toes is virtually identical with one showing the distribution of valgus deviation of the great toe in normal young adults (Hardy and Clapham 1951); thus it is reasonable to conclude that the deviated and subluxated toes show the same amount of lateral tilting of the articular surfaces as is found in normal feet, whereas the congruous group covers only the upper half of the normal range.

Nature of the congruous type of hallux valgus—The above considerations clearly suggest that the congruous type represents merely the upper range of the variable valgus alignment of the normal great toe, and, unlike the deviated and subluxated toes, it is not a pathological condition. This view is substantiated by the following observations: 1) the radiologically measured hallux valgus in this group ranged from 15 to 28 degrees, which is within the normal range established by Hardy and Clapham (1951) and by Harris and Beath (1947); the same applied to the

intermetatarsal angle, though the mode of both these values was slightly higher than normal as would be expected if we are dealing with the upper range of normal. The " normal range " from any physiological or anatomical value is of course a terminological conception rather than a natural phenomenon, but the essential fact is that no patient with this type showed a greater degree of hallux valgus than may be found in any group of young people who have never been troubled by their feet, consider themselves to be quite normal, show no associated abnormality, give no family history of hallux valgus, and are otherwise unselected—which is the definition of " normal " used by the above-quoted authors. 2) The average age of the



CONGRUOUS TYPE — FIG. 6

DEVIATED TYPE — FIG. 7

SUBLUXATED TYPE — FIG. 8

Tracings of radiographs showing the different ways in which increased valgus of the great toe may occur. In Figure 6 the articular surfaces are set more obliquely than usual to the long axes of their supporting bones, but their relationship to each other is normal, and the anatomical variation is in the shape of the bones. In Figure 7 the bones are normal in shape, but the articular surfaces are not congruous with each other, so increased valgus occurs at the joint. Figure 8 is a further stage of that in Figure 7. These three examples show approximately the same degree of hallux valgus, 25, 23 and 22 degrees respectively.

patients in the congruous group did not differ significantly from that of the patients in the deviated and subluxated groups; thus there is no justification for regarding this group as an earlier stage of the other two (in fact the patients with congruous joints were on average slightly *older* than the others). 3) No patient in the congruous group showed rotation of the great toe, displacement of the sesamoids, or any other abnormality of the forefoot. 4) There were only twenty feet in this group observed over periods varying from two to fifteen years (average six years nine months) and it would therefore be unwise to make any firm statement about their natural evolution; but such limited observations as could be made were in complete agreement with the view that these were normal feet, because no patient developed either deviation or subluxation, and over the group as a whole the degree of hallux valgus and the intermetatarsal angle remained constant with age. In other words there was no suggestion of progressive deformity in these feet, but the numbers seen were too small and the periods of observation too short for it to be said quite definitely that it never occurred.

Relationship of the deviated and subluxated groups to each other—The following evidence leaves little doubt that deviation and subluxation represent different stages of the same process of lateral displacement of the proximal phalanx, though progression from one to the other by

no means always occurs. 1) Eight feet in the deviated group were seen to develop subluxation during the period of observation covered by this study, which ranged from one to twenty-one years in individual patients (Figs. 10 and 11). It was not necessarily the oldest, nor those who had been observed for the longest period, who showed this change, for these eight patients were evenly scattered throughout the various age groups, and the toe of the patient who was observed for twenty-one years was still of the deviated type at the end of that time. 2) There were eighty-one feet in the deviated group and 114 in the subluxated. These were distributed among 100 patients and, though as a general rule both feet in any given case showed the same type of deformity, twenty-three had deviation on one side and subluxation on the other, again demonstrating a close association of the two groups. 3) Hallux valgus in the deviated group

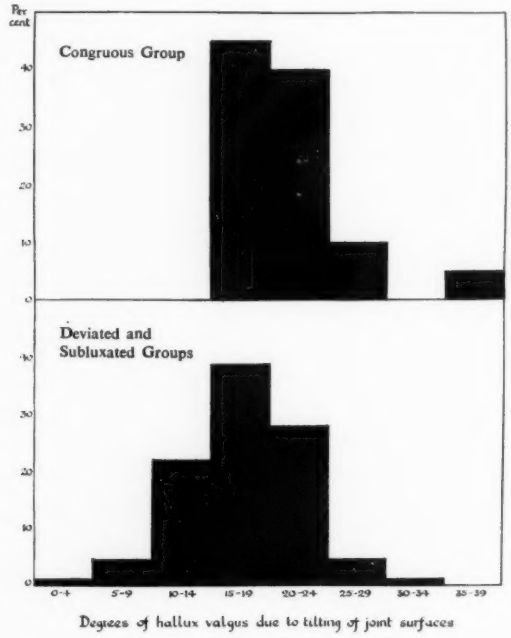


FIG. 9

Showing that the combined lateral tilting of the articular surfaces—that is, the " bony component " of the valgus—ranges from 0 to 34 degrees in the deviated and subluxated groups, but in the congruous group it covers only the upper part of this range. The deviated and subluxated groups separately give figures similar to the combined one shown here.

ranged from 15 to 35 degrees with a mode of 25 degrees, whereas in the subluxated group the figures were higher, the range being 19 to 60 degrees and the mode 30 degrees. Similarly, the intermetatarsal angle was smaller in the deviated than in the subluxated toes, the ranges being 5 to 17 degrees in the former, and 7 to 20 degrees in the latter, with modes of 11 and 14 degrees respectively. These findings merely show that the deformity is more marked in the subluxated group than in the deviated, which is to be expected if the two types are different stages of the same condition.

Structural prognosis of the deviated and subluxated toes—To give a comprehensive and unarguable account of the structural prognosis of adolescent hallux valgus would require the observation of large numbers of untreated cases for forty years or more. However, from this

much more limited study certain pointers have emerged which strongly suggest that, in the absence of subluxation, progression of the deformity is slight or absent. 1) Figure 12 shows that, when each group is considered as a whole, the degree of hallux valgus increases with age in the subluxated, but not in the deviated type. Figure 13 shows similar features for the intermetatarsal angle but, as this is considerably smaller than the angle of hallux valgus, the difference is not so immediately obvious. 2) It is however only by observing individual patients that we can obtain indisputable evidence of progression. The natural evolution of hallux valgus is a very gradual process, and measurement of consecutive standardised radiographs is the only reasonably accurate way of recording it; therefore only those feet which have such films covering periods of two years or more will be considered here. The longest period of

FIG. 10 FIG. 11

Two radiographs of the same foot with an interval of six and a half years. Figure 10, taken when the patient was twelve years old, shows deviation of the proximal phalanx on the metatarsal head; Figure 11, at the age of eighteen, shows early subluxation.

standardised radiological observation so far is six and a half years. As stated above, the standard error of angular measurement is ± 1 degree, and so an increase of more than 2 degrees has been taken to indicate progression; clearly, therefore, some slowly progressive cases will be " missed," but this factor will be common to both groups and will not affect comparisons between the two.

Under these criteria there were twenty-four feet in the deviated group with the necessary radiographs, and, of these, nine showed an increase in the angle of hallux valgus. This evidence of progression in individual cases may at first sight conflict with the lack of it in the group as a whole (Figs. 12 and 13), until it is recalled that some feet of this type develop subluxation and therefore do not appear in the scattergraphs of the deviated group; it seems therefore that increase of hallux valgus occurs in some of the deviated type, and soon results in subluxation. In the group with subluxation, out of twenty-seven feet with the necessary radiographs thirteen showed an increase in the degree of hallux valgus.

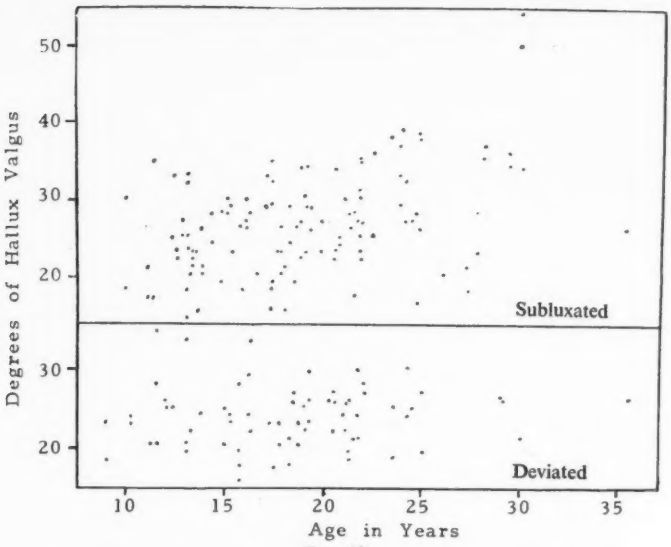


FIG. 12

Scattergraph showing that in the subluxated group the degree of hallux valgus increases with age; the deviated group shows no evidence of such progression. Each dot represents one foot.

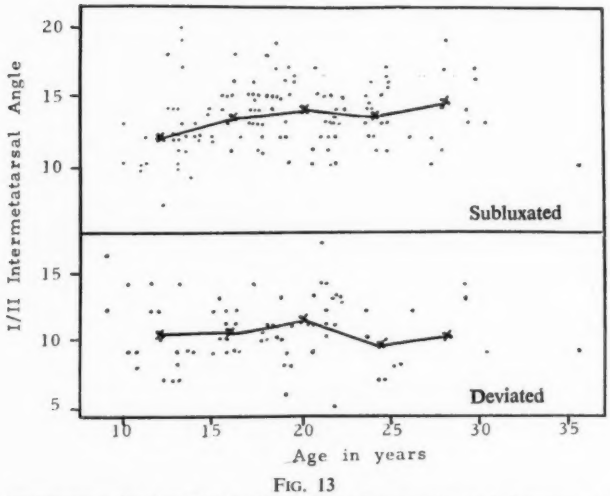


FIG. 13

Scattergraph showing that in the subluxated group the angle between the first and second metatarsals increases with age; the deviated group shows no evidence of such progression. The x joined by a continuous line represents the mean values. Each dot represents one foot.

FIG. 14 FIG. 15 FIG. 16

Tangential views of the forefoot to show the sesamoid bones. In Figure 14 they are in the normal position with a well-marked interposing ridge on the metatarsal head. In Figure 15 there is some lateral displacement with moulding of the ridge. Figure 16 shows more marked lateral displacement and the ridge is flattened.

No foot in the group with deviation showed measurable change in the intermetatarsal angle, but an increase was observed in four feet of the subluxated type. It should be noted that, since the standard error of measurement was the same for hallux valgus and for the intermetatarsal angle, and since the actual angle in the latter was much smaller, one would expect to find a measurable increase less commonly in the intermetatarsal angle than in the amount of hallux valgus.

Rotation of the great toe—Rotation of the great toe about its longitudinal axis was not seen in the group with congruous joints. Nine feet (11 per cent) in the deviated group and forty-three (38 per cent) in the subluxated group showed rotation varying from 5 to 45 degrees as estimated approximately by sight.

Displacement of the sesamoids—The lateral displacement of the sesamoids which occurs in hallux valgus has been well described by Haines and McDougall (1954). This shift can be seen in ordinary antero-posterior radiographs (Figs. 2 to 4), but is best shown by the sesamoid view illustrated in Figures 14 to 16; moulding of the intersesamoid ridge on the metatarsal head precedes the actual displacement of these bones. In all the feet with congruous joints the sesamoid view was normal; eleven (14 per cent) of the deviated toes, and sixty-four (56 per cent) of the subluxated toes showed displacement of variable extent corresponding approximately with the degree of hallux valgus. As might be expected rotation of the great toe was nearly always associated with sesamoid displacement, though displacement of the sesamoids occurred fairly frequently in the absence of rotation.

Severity of symptoms—Symptoms are notoriously difficult to assess, but a classification based on the degree of restriction of normal activities was made as follows: 1) no pain or discomfort at any time; 2) mild—some discomfort but no curtailment of activity; 3) moderate—some restrictions such as avoiding dancing or long walks; 4) severe—occasional complete cessation of normal activity involving rest in bed or staying off work. It was found that none of the patients with congruous joints had pain; sixteen (20 per cent) of the patients with deviated toes had mild symptoms, four (5 per cent) moderate and one (1 per cent) severe; the figures for the subluxated group were thirty-two (28 per cent) mild, fifteen (13 per cent) moderate and one (1 per cent) severe.

Family history—Family history is mentioned only to stress its notorious fallaciousness; very few patients were well informed about their parents' feet and still fewer about their grandparents'; also it happened on several occasions that the mother accompanying a patient insisted that her own feet were completely normal even after the reluctant removal of her shoes had revealed quite marked deformity.

DISCUSSION

Structural prognosis—It is suggested above that pathological hallux valgus (deviation and subluxation) can be distinguished from an exaggeration of the normal valgus alignment of the

great toe (congruous type) by the relationship of the articular surfaces of the first metata o-phalangeal joint to each other; no arbitrary angle can be given above which hallux valg is pathological and below which it is normal, for in the congruous—or " exaggerated norma — group it reached as high as 28 degrees, whereas subluxation was observed with a valgus defor ity of only 19 degrees and deviation with valgus of only 15 degrees. To recapitulate, those join in which the articular surfaces are congruous and parall re regarded as representing the upper range of normal valgu e-cause they show no definite pathological change but mere an exaggeration of a normal feature, and over the period co red by this study they showed no sign of developing progr ive deformity; it has not so far been possible to follow eir individual progress beyond the age of thirty-six years, an t is of course possible that some may develop degenerative ch ges in the joint in later life, but in 100 consecutive adults, ho attended because of symptoms, this picture was not en. It therefore seems clear that patients in this small g up require no treatment in adolescence, and can be reass red about their future with some degree of confidence.

FIG. 17

Hallux valgus in a sixty-year-old woman with pain over the metatarsal head and early degenerative changes in the first metatarso-phalangeal joint. Note deviation of phalanx but no subluxation.

In the group with subluxated joints progression of the deformity is common; as shown above, over the relat ely short periods covered by standardised radiological obs va-tion, about 50 per cent of this group showed signific ntly measurable progression and this figure, of course, repres nts the absolute minimum, the true proportion of progressive cases probably being much higher. The periods of observation of individual cases have not been long enough to indicate whether *all* deteriorate, but it seems clear that many of this type will develop into the well known disorganised forefeet of later adult life.

The position in the group with deviated toes is not so clear. Some certainly progress to subluxation quite quickly, and for these the considerations already outlined for the subluxated feet apply; others in this series have remained deviated without subluxation for as long as twenty-one years, and the fact that the radiological appearances shown in Figure 17 are a fairly common finding in middle age and later suggests that in many of this type subluxation never does occur, though degenerative joint changes may do so.

To summarise the structural prognosis of hallux valgus in adolescents, congruity of the joint surfaces can be regarded as normal and indicating that progressive deformity will not occur; subluxation indicates that deterioration is likely; with deviation it is necessary to wait and see, as some of this type develop subluxation and then become progressive whereas others remain unchanged.

The role of " metatarsus primus varus "—It is not proposed to go into details on the highly controversial matter of treatment of hallux valgus in its early stages, but as many of the corrective operations in use aim at decreasing the angle between the first and second metatars als (Lapidus 1934, Ellis 1951) it is pertinent to inquire just what part this " metatarsus prim us varus " plays in the production of hallux valgus. A search of all the available English literat ure has revealed that, although the idea that metatarsus primus varus is the underlying ca use of hallux valgus is widely held, little or no evidence has been produced to support it; moreov r, most authors are not clear as to how medial deviation of the first metatarsal develo s, some implying that it is a congenital or growth anomaly (Truslow 1925, Jones 194).

other suggesting that it is essentially a spreading of the forefoot caused by weight bearing (Mizuho, Sima and Yamazaki 1956).

the primary lesion were medial deviation of the metatarsal from any cause, it is easy to see how lateral deviation of the great toe would develop; the base of the proximal phalanx is connected very firmly indeed to the sesamoids, and through them to the intermetatarsal ligament, so that as the metatarsal moved medially the phalanx, being held laterally, would glide round the head of that bone into the valgus position. The consequent bowstringing of the long extensor and flexor tendons across the lateral side of the joint, and the altered line of pull of adductor hallucis, the lateral head of flexor hallucis brevis, and extensor hallucis brevis would then perpetuate and increase the deformity. But this perpetuating mechanism of altered muscle alignment would apply in exactly the same way if lateral deviation of the phalanx were the primary change; furthermore, these muscles would tend to pull the lateral side of the base of the proximal phalanx into the space between the first and second metatarsal heads, widening the anterior end of this space and thus, particularly after subluxation had occurred, producing medial deviation of the first metatarsal as a secondary change.

This question of whether lateral deviation of the proximal phalanx or medial deviation of the metatarsal develops first is of more than academic interest because it seems unlikely that a true permanent correction of the deformity will be obtained unless the primary condition is corrected. The present study cannot claim to give a definite answer to this problem, which is not unlike that of the chicken and the egg, but one relevant fact can be established: when the subluxated group is considered as a whole, both the intermetatarsal angle and the amount of hallux valgus increased with age, and this was confirmed in the smaller number of individual cases for whom consecutive standardised radiographs were available. The very high intermetatarsal angles found in older patients (up to 27 degrees) were not seen in the younger ones studied here and it therefore seems that hallux valgus and medial deviation of the first metatarsal increase *pari passu*, in some cases throughout life; that is to say that there is a continuous progressive buckling of the first skeletal ray and there is no support for the conception of a fixed congenital or growth anomaly of the first metatarsal.

Less conclusively, this study supports the view of Hardy and Clapham (1952) and McMurray (1936) that lateral deviation of the phalanx is the primary change in hallux valgus, although it throws no light on the cause of this deviation. Subluxation of the first metatarso-phalangeal joint is a very early phenomenon: it was seen in forty-one feet before the metatarsal and phalangeal epiphyses were closed, the youngest patient being nine and a half years old; altogether 114 (53 per cent) of the feet examined showed subluxation when first radiographed, whereas only five (2·3 per cent) showed an intermetatarsal angle beyond the normal range. This suggests that subluxation is a commoner early change than is an increase in the intermetatarsal angle, but evidence of this nature is open to the serious criticism that in any foot an intermetatarsal angle well within the normal range may (and frequently does) represent an increase above the normal for that particular foot; however, this criticism applies with equal force to the measurement of hallux valgus as well as the intermetatarsal angle, and on assessment twenty-six feet were found to have hallux valgus beyond the upper limit of normal compared to five with a similar increase in the intermetatarsal angle. To sum up, it can be said that subluxation, the abnormality of which is beyond doubt, is a very frequent early change; it can occur with an intermetatarsal angle as low as 7 degrees, and is a much more common finding in young patients than medial deviation of the first metatarsal so marked as to be unequivocally abnormal; it therefore seems likely that the so-called metatarsus primus varus is secondary to lateral displacement of the proximal phalanx.

SUMMARY

Pathological hallux valgus may be differentiated from an increase in the normal valgus alignment of the great toe by the relationship to each other of the articular surfaces of the

first metatarso-phalangeal joint; these are congruous in the normal joint, but displaced on each other in the pathological.

2. The earliest change is lateral deviation of the proximal phalanx on the metatarsal head, which may progress rapidly to subluxation.

3. Subluxation is an early change in a high proportion of cases, and is frequently present when the patient is first seen in adolescence.

4. Once subluxation has occurred progression of the deformity is likely.

5. Metatarsus primus varus and hallux valgus increase *pari passu*. There is not enough evidence to be certain which of these is the primary change, but such as there is suggests that lateral deviation of the great toe occurs first, and increase in the intermetatarsal angle is secondary to this.

6. It is suggested that hallux valgus should be regarded primarily and fundamentally as a subluxation, or tendency to subluxation, of the first metatarso-phalangeal joint.

It is a pleasure to express my thanks to the surgical staff of the Royal National Orthopaedic Hospital for permission to examine their patients, and in particular to Mr A. T. Fripp for constant advice and encouragement during the preparation of this paper; and gratefully to acknowledge ready assistance received from the staffs of the Radiographic and Medical Records Departments of the Royal National Orthopaedic Hospital and the Department of Medical Photography of the Institute of Orthopaedics.

REFERENCES

BONNEY, G., and MACNAB, I. (1952): Hallux Valgus and Hallux Rigidus. A Critical Survey of Operative Results, *Journal of Bone and Joint Surgery*, **34–B,** 366.

ELLIS, V. H. (1951): A Method of Correcting Metatarsus Primus Varus. Preliminary Report. *Journal of Bone and Joint Surgery*, **33–B,** 415.

HAINES, R. W., and MCDOUGALL, A. (1954): The Anatomy of Hallux Valgus. *Journal of Bone and Joint Surgery*, **36–B,** 272.

HARDY, R. H., and CLAPHAM, J. C. R. (1951): Observations on Hallux Valgus. Based on a Controlled Series. *Journal of Bone and Joint Surgery*, **33–B,** 376.

HARDY, R. H., and CLAPHAM, J. C. R. (1952): Hallux Valgus. Predisposing Anatomical Causes. *Lancet*, **i,** 1,180.

HARRIS, R. I., and BEATH, T. (1947): *Army Foot Survey*. Ottawa: National Research Council of Canada.

JONES, A. Rocyn (1948): Hallux Valgus in the Adolescent. *Proceedings of the Royal Society of Medicine* (Section of Orthopaedics), **41,** 392.

LAPIDUS, P. W. (1934): Operative Correction of the Metatarsus Varus Primus in Hallux Valgus. *Surgery, Gynecology and Obstetrics*, **58,** 183.

MCMURRAY, T. P. (1936): Treatment of Hallux Valgus and Rigidus. *British Medical Journal*, **ii,** 218.

MIZUNO, S., SIMA, Y., and YAMAZAKI, K. (1956): Detorsion Osteotomy of the First Metatarsal Bone in Hallux Valgus. *Journal of the Japanese Orthopaedic Surgical Society*, **30,** 105.

TRUSLOW, W. (1925): Metatarsus Primus Varus or Hallux Valgus? *Journal of Bone and Joint Surgery*, **7,** 98.

HALLUX VALGUS IN ADOLESCENTS

F. A. SIMMONDS and M. B. MENELAUS, PYRFORD, ENGLAND

From the Rowley Bristow Orthopaedic Hospital, Pyrford

Hallux valgus is associated with metatarsus varus, and while the foot is growing the deformity usually progresses. This article describes the results of a reconstructive operation which is designed to correct both the components of the deformity and to maintain the correction. It has been performed thirty-three times in six years on children between the ages of nine and eighteen years, and the results are sufficiently encouraging to permit us to advocate its use in certain circumstances. Metatarsus primus varus might more correctly be referred to as metatarsus primus adductus, but as the former term is generally used it will be retained in this article.

Pointing out the futility of conservative measures in the treatment of hallux valgus, Cholmeley (1958) stated that if correction of the great toe deformity is necessary in childhood or adolescence, operation is the only way to achieve it. McBride (1954) considered it unfortunate that hallux valgus was not more often recognised and corrected in the formative stage, before permanent articular and ligamentous changes had occurred. He described an operation (McBride 1928, 1954) involving transfer of the adductor hallucis and reconstruction of the metatarso-phalangeal joint of the hallux. These procedures form part of the operation reviewed in this article.

In a review of 518 operations of all types for hallux valgus Bonney and Macnab (1952) found that the best results in the entire series were achieved in those adolescents in whom a permanent correction of the metatarsus primus varus had been achieved. They noted that it was difficult to obtain this permanent correction. Whether or not the varus deformity of the first metatarsal is the primary factor in the development of hallux valgus, it certainly leads to a " breakdown of the mechanical integrity of the forefoot and often causes more pain and dysfunction than the valgus deformity of the great toe " (Stamm 1957).

To obtain and hold correction of the metatarsus varus an osteotomy of the base of the first metatarsal is advocated in addition to McBride's soft-tissue reconstruction. A similar combined operation was described by Stamm (1957), but most of the patients were older (Evans 1957) and, in addition, excision of the base of the phalanx was often performed.

The operation described below was devised by one of us (F. A. S.) and has been performed at the Rowley Bristow Orthopaedic Hospital since 1952. Table I indicates the ages at which it has been performed and the length of the follow-up. Operations in the last year have not been included in this series.

INDICATIONS FOR OPERATION

The indications for operation are: 1) Hallux valgus deformity sufficiently severe to make the foot ugly, to cause difficulty in obtaining comfortable shoes, or to cause pain. In this series symptoms of first metatarsal head pressure were present in twenty-four feet. 2) Hallux valgus deformity likely to become severe. If serial radiographs, taken at intervals of six months, show progressive increase in an initially mild deformity, it is likely to become severe. A further indication that the valgus deformity is likely to increase is the presence of a family history. Bonney and Macnab (1952) also pointed this out.

Although there is no statistical evidence, our impression is that the results of operation in older children are less satisfactory than in those operated upon before the foot is fully grown. We now consider that the most satisfactory age for operation is between eleven and fifteen years. It is possible that operation before the age of eleven might result in premature fusion of the epiphyses and a short great toe.

The indications for this operation are not commonly satisfied and there is a natural reluctance to operate on symptomless feet. This is shown by the fact that the operation has been performed on only thirty-three occasions between 1952 and 1958 on patients from the out-patient clinics of the Rowley Bristow Orthopaedic Hospital and its associated children's clinics, and St Thomas's Hospital.

The operation is not indicated if there is a poor range of dorsiflexion of the metatarso-phalangeal joint of the hallux.

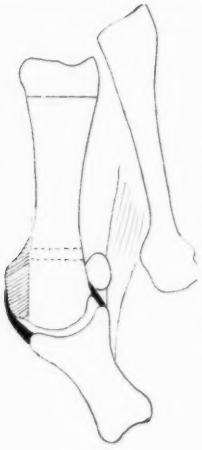


FIG. 1

FIGS. 1 AND 2

The anatomy before and after operation of the first and second metatarsals and the proximal phalanx of the hallux. In the first diagram the shaded part of the metatarsal head represents the knob to be excised. The dotted lines in the metatarsal neck indicate the drill hole, and the site of the osteotomy at the base of the metatarsal is shown. The second diagram illustrates the arrangement after operation; the lateral capsule has been divided and the medial capsule tightened. The adductor tendon has been threaded through and a wedge osteotomy performed.

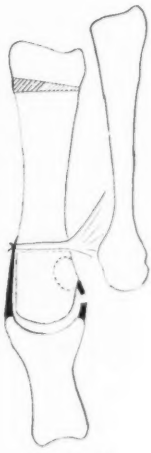


FIG. 2

TECHNIQUE OF OPERATION

The operation is performed under tourniquet through two incisions, a long dorso-medial incision extending from the base of the first metatarsal to the middle of the proximal phalanx of the hallux, and a short incision just lateral to the first metatarso-phalangeal joint. Through the medial incision the medial ligament of the metatarso-phalangeal joint is defined, divided close to its metatarsal attachment and reflected. The underlying prominent knob of bone is removed with an osteotome (Figs. 1 and 2) and put aside. Through the lateral incision the tendon of the adductor hallucis is defined and detached from the proximal phalanx and from the lateral sesamoid bone. This tendon is then threaded through a transverse drill hole in the first metatarsal neck to be sutured later. The lateral capsule of the metatarso-phalangeal joint is divided. The first metatarsal bone is divided transversely half an inch distal to its base, and the bone previously removed from the metatarsal head is shaped into a wedge and driven in between the divided bone fragments; the shaft is thereby forced into a less varus position and the osteotomy is then completely stable. With the hallux held in a varus position, the medial capsule is sutured with catgut mattress stitches placed so that the capsule is shortened; the end of the adductor tendon is sutured to the medial capsule. The shortened medial capsule should hold the toe in about 10 degrees of overcorrection. The incisions are closed in two layers and a plaster toe cap, extending from the tuberosity of the navicular bone to the tips of the toes, is applied.

The operation described above was performed, with minor variations, in thirty of the thirty-three operations reviewed. In three of these thirty operations a single curved incision replaced the two separate incisions, but this was unsatisfactory in two because of delayed healing of the wound. In three of the thirty-three operations the adductor tendon was divided, but not threaded through the neck of the metatarsal bone. In some patients when the osteotomy was performed, an attempt was made to arrest the growth of the lateral part of the epiphysis

of the first metatarsal bone by directing the osteotome obliquely into it. This modification was abandoned because it made the osteotomy unstable.

Management after operation—The plaster toe cap is retained for two weeks; then the sutures are removed and a below-knee walking plaster, extending to the tips of the toes, is applied. A loop of plaster across the first web holds the hallux in its corrected position. The patient is discharged walking in a plaster boot. After four weeks the plaster is removed and radiographs are taken. The patient is encouraged to exercise the toe and to walk with a normal gait. Most patients are readmitted for a few days for this final removal of plaster and for physiotherapy.

Stiffness of the metatarso-phalangeal joint was noted after operation in several cases: usually movement slowly improved with exercise, but in four patients some degree of rigidity of the toes persisted up to the time of review. Some patients treated at the beginning of the series were given a night splint to prevent the toe from returning to a valgus position in the first few months after operation.

RESULTS

The operation was performed on thirty-three occasions on the feet of twenty-three girls between the ages of nine and eighteen. Ten patients had bilateral and thirteen patients unilateral operations. Of the twenty-three patients, twenty-two have been reviewed by one of us (M.B.M.). The remaining patient has been reviewed recently by a surgeon in New Zealand and he has sent the information to us. Table I indicates the time between operation and review.

At review, the presence of any symptoms, the patient's impression of the results of the operation, the appearance of the foot and the range of movement at the metatarso-phalangeal joint were recorded. A radiograph and, in most cases, a photograph of both feet, were taken. The radiographs were taken with the patient standing on the casette with the x-ray tube vertically above a point midway between the first metatarsal heads, the target-film distance being three feet. The angle between the long axis of the first metatarsal bone and the long axis of the proximal phalanx of the great toe, and the angle between the first and second metatarsal bones (intermetatarsal angle) were measured on the standing radiographs and compared with the angle before operation (except in six cases in which radiographs taken before operation were not available; in these patients photographs and clinical notes have had to suffice for making comparisons).

Patient's assessment—Table II shows that thirty-one operations satisfied the patient and the parent. Patients tend to be pleased with results that do not necessarily satisfy the surgeon from the functional and anatomical viewpoints, although the cosmetic appearance is important.

Twenty feet were completely asymptomatic at the time of review. Six feet were admitted to be the site of occasional aching in the region of the big toe joint when the patient was dancing, at the end of a hot day or in cold weather. In these, the ache was less than had been present before operation. Six feet had the same amount of aching as before operation and the one dissatisfied patient complained of increased pain. This girl had cavus feet and probably should not have had the operation.

Effectiveness of the operation in correcting deformity—Table III shows that in twenty-six feet a correction of the hallux valgus of more than 10 degrees had been maintained up to the time of review. Although the correction of 10–20 degrees obtained in thirteen feet does not, at first sight, appear satisfactory, it must be remembered that the valgus before operation in adolescents is much smaller than that usually seen in adults: the average angle was, in fact, 21 degrees and in four feet the valgus before operation was less than 20 degrees. Furthermore, the prime indication for operation was progressive hallux valgus and the operation was performed as a prophylactic measure and always on the worst side first. The review showed that, of the thirteen patients who had operation on one side, eleven showed a greater degree of valgus on the untreated side at the time of review (Table IV). Although hallux valgus may be confined to one side, Table V shows that there was a tendency for the deformity to progress

TABLE I

AGE AT TIME OF OPERATION AND LENGTH OF FOLLOW-UP
(Twenty-three patients; thirty-three operations)

Age at operation	Number of operations
9 to 10 years	2
10 to 12 years	8
12 to 14 years	8
14 to 16 years	13
18 years	2
Length of follow-up	
1 to 3 years	20
3 to 6 years	11
Over 6 years	2

TABLE II

PATIENTS' AND PARENTS' ASSESSMENT OF THE RESULTS
(Thirty-three operations)

Satisfied	Not satisfied	Uncertain
31	1	1

TABLE III

COMPARISON OF THE DEGREE OF VALGUS BEFORE OPERATION AND AT REVIEW

More than 20 degrees of correction of hallux valgus maintained	10 to 20 degrees of correction maintained	Less than 10 degrees of correction maintained	Hallux valgus greater than before operation
13 feet	13 feet	4 feet	3 feet

TABLE IV

COMPARISON OF HALLUX VALGUS ON SIDE OPERATED UPON AND ON UNTREATED SIDE AT TIME OF REVIEW

Side operated upon less valgus than untreated side	Side operated upon more valgus than untreated side
11 feet	2 feet

TABLE V

PROGRESS OF THE HALLUX VALGUS ON THE UNTREATED SIDE IN PATIENTS WHO ORIGINALLY HAD UNILATERAL OPERATION

Hallux valgus progressed	Hallux valgus unchanged	Hallux valgus improved
11 feet (3 of these patients have undergone operation and 1 is awaiting operation)	4 feet	1 foot

TABLE VI

COMPARISON OF INTERMETATARSAL ANGLES BEFORE OPERATION, AFTER OPERATION AND AT REVIEW

Case number	Side of operation	Age at operation (*years*)	Angle before operation (*degrees*)	Angle after operation (*degrees*)	Angle at review (*degrees*)
1	L	12	13	(Films not available)	4
2	L	10	11	7	6
	R	10	11	2	2
3	L	10	18	2	2
4	R	12	16	5	4
5	L	13	10	4	3
	R	13	11	4	4
6	L	14	15	10	15
	R	14	16	11	11
7	R	10	(Films not available)	6	5
8	L	9	(Films not available)	8	7
9	L	15	24	7	9
10	L	14	8	Parallel	2
	R	14	8	Parallel	Parallel
11	L	14	(Films not available)	(Films not available)	8
12	L	14	12	4	4
13	L	13	19	12	12
14	L	11	9	4	4
	R	12	7	7	5
15	L	15	(Films not available)	8	7
16	L	9	15	8	5
17	L	15	16	(Films not available)	7
	R	13	15	9	7
18	L	12	9	(Films not available)	4
	R	12	8	(Films not available)	4
19	L	14	(Films not available)	6	6
20	L	15	15	3	3
	R	15	11	4	7
21	L	14	15	9	8
22	L	18	(Films not available)	4	4
	R	18	(Films not available)	8	10
23	L	11	10	7	7
	R	11	12	8	5

on the untreated side, and it is reasonable to assume that the deformity would also have progressed on the side operated upon.

Tables VI and VII show the effectiveness of the operation in correcting the metatarsus varus deformity. It must be remembered that the normal intermetatarsal angle in children between four and fifteen years of age is about 7 degrees (Hardy and Clapham 1952); in this series the average angle before operation was 13 degrees.

TABLE VII

COMPARISON OF INTERMETATARSAL ANGLES BEFORE OPERATION AND AT REVIEW

More than 8 degrees of correction	4 to 8 degrees of correction	0 to 4 degrees of correction
11 feet	12 feet	3 feet

TABLE VIII

RANGE OF DORSIFLEXION AT METATARSO-PHALANGEAL JOINT ON TREATED SIDE AT TIME OF REVIEW

More than 45 degrees of dorsiflexion	15 to 45 degrees of dorsiflexion	Less than 15 degrees of dorsiflexion
22 feet	7 feet	4 feet

Effectiveness of the operation in maintaining correction—The correction of the metatarsus varus obtained at operation was generally maintained. Table VI shows that a few degrees of correction were lost in only six feet.

No figures can be given to show how correction of the valgus deformity of the toe was maintained, because at the time of the radiographs taken after operation the toes were still in the overcorrected position in which they had been splinted to relax the medial ligament. The toe always swings back to some extent from this varus position. Nevertheless, Table III shows that a satisfactory correction was generally present at the time of review.

It is difficult to draw any conclusions with regard to the effect of the operation on epiphysial fusion. There is no evidence that the attempts made to destroy the lateral part of the epiphysial line were successful in causing early fusion of this part of the epiphysis. In six of the thirteen unilateral operations radiographs demonstrated accelerated union of the epiphysis on the affected side. In three of these six instances the metatarsal epiphysis, and in three the epiphysis of the proximal phalanx, fused earlier on the affected side. These numbers are too small to be significant, but are of interest and suggest that the operation should not be performed at too early an age.

Range of dorsiflexion at the metatarso-phalangeal joint—It is important that operation should not produce a stiff metatarso-phalangeal joint. Table VIII shows the range of passive dorsiflexion at this joint at the time of review. None of the patients complained of stiffness of the joint or of inability to wear high-heeled shoes; some may do so when they are older. The Table shows considerable variation in the range of dorsiflexion in the treated feet but there is a wide range of variation in normal feet (Joseph 1954). In the thirteen patients in whom operation was performed on one side only this range varied from 30 degrees to 90 degrees on the untreated side and in six of these dorsiflexion of the hallux operated upon was equal to, or greater than, that on the side not requiring surgery. The movement was a little poorer on the treated side in the remaining seven patients, but in only two of these was dorsiflexion diminished by as much as 30 degrees.

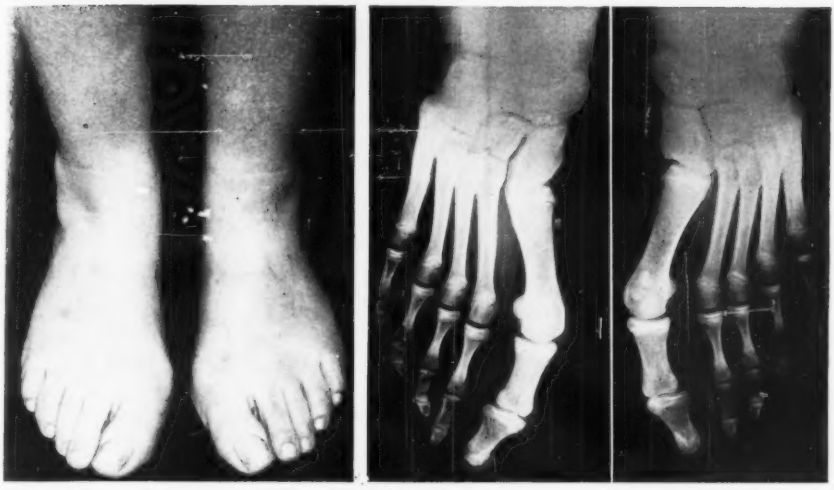


FIG. 3

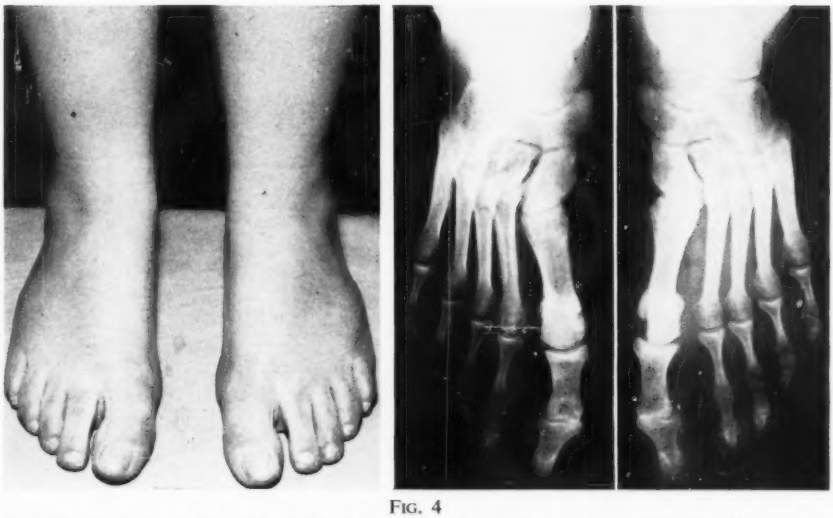


FIG. 4

Case 14—Photographs and radiographs of both feet taken before operation (Fig. 3) and at review (Fig. 4). The right foot was operated upon four years and seven months before the review photograph and the left foot seven months later. The patient was eleven and a half years old at the time of the first operation.

General assessment—The results were considered good in twenty-six feet in that the hallux valgus was well corrected, the foot was of good appearance and the patient was satisfied (Figs. 3 and 4).

The results were considered to be moderate in four instances because the correction of the hallux valgus obtained was insufficient, or the patient had symptoms which were not improved by the operation. These patients were, nevertheless, pleased that they had had the operation.

In three feet the result was poor in that the hallux valgus was not corrected, and one of these patients had increased pain. This was the girl, previously mentioned, with the cavus foot.

DISCUSSION AND CONCLUSIONS

Bonney and Macnab (1952) suggested that one of the causes of failure to mai tin correction of metatarsus varus was that the first metatarsal was divided distal to its gro ng epiphysis. They considered that further bone growth occurring in a varus direction caused rly recurrence of the deformity. The results of the thirty-three operations considered in his article do not confirm their suggestion. In fact, we now prefer to operate on children u ler fifteen years of age. The reasons for this preference are that the correction of hallux va us and metatarsus varus is at least as well maintained in these as in older children and that t ere does not appear to be as high an incidence of stiffness of the metatarso-phalangeal joi if operation is performed early. For example, after the eleven operations performed in chil en under the age of twelve the range of dorsiflexion of the metatarso-phalangeal joint was r er less than 15 degrees when the patients were reviewed. Moreover, of the children who under nt operation under the age of fourteen, a higher proportion had a " good " result than an ng those operated upon after that age. The number of patients treated is too small to ind ate whether simple division of the adductor hallucis tendon is as effective as threading the div led tendon through the metatarsal neck. In the three instances in which the tendon was div led two results were classed as " good " and one as " moderate." We are at present investiga ing whether this simpler procedure is as effective by performing it on one of each pair of feet requiring surgery.

McBride (1954) considered that it was important to excise the lateral sesamoid bone to allow adequate approximation of the first and second metatarsal heads. We have not interfered with the sesamoid bones and our results suggest that it is not necessary to do so in adolescents.

Because in two of the three operations in which primary wound healing did not occur a single curved incision was employed, this incision, which has been used on only three occasions, is not advised.

SUMMARY

1. An operation suitable for the correction of hallux valgus in adolescents is described.
2. The results of thirty-three operations performed in children between the ages of nine and eighteen years are analysed.
3. Twenty-six operations are considered to have produced a good result, four a moderate result, and three a poor result.
4. It is suggested that the operation is best performed between the ages of eleven and fifteen years.

We are grateful to Professor George Perkins and Mr A. Graham Apley for permission to review several patients in this series who were under their care.

REFERENCES

BONNEY, G., and MACNAB, I. (1952): Hallux Valgus and Hallux Rigidus; A Critical Survey of Operative Results. *Journal of Bone and Joint Surgery*, **34-B,** 366.

CHOLMELEY, J. A. (1958): Hallux Valgus in Adolescents. *Proceedings of the Royal Society of Medicine* (Section of Orthopaedics), **51,** 903.

EVANS, D. K. (1957): The Operative Treatment of Hallux Valgus. A Review of a " Radical " Operation. *Guy's Hospital Reports*, **106,** 280.

HARDY, R. H., and CLAPHAM, J. C. R. (1952): Hallux Valgus. Predisposing Anatomical Causes. *Lancet*, **i,** 1180.

JOSEPH, J. (1954): Range of Movement of the Great Toe in Men. *Journal of Bone and Joint Surgery*, **36-B,** 450.

MCBRIDE, E. D. (1928): A Conservative Operation for Bunions. *Journal of Bone and Joint Surgery*, **10,** 735.

MCBRIDE, E. D. (1954): Hallux Valgus, Bunion Deformity: its Treatment in Mild, Moderate and Severe Stages. *Journal of the International College of Surgeons*, **21,** 99.

STAMM, T. T. (1957): The Surgical Treatment of Hallux Valgus. *Guy's Hospital Reports*, **106,** 273.

NEUROPATHIC JOINTS IN DIABETES

DONALD P. DEGENHARDT and MARTIN A. GOODWIN, DARLINGTON, ENGLAND

From the Darlington Memorial Hospital

It is well recognised that neuropathy is common in diabetes mellitus, not only in the elderly, but also in younger patients with poorly controlled diabetes of long duration. It is not so clearly recognised that some patients with advanced lesions will develop neuropathic changes in the joints. Jordan (1936) seems to have been the first to have recognised this, and Bailey and Root (1947) found seventeen such patients in 20,000 diabetics. Out of 150 patients with diabetic neuropathy Martin (1953) discovered nine with " Charcot " joints.

Usually affecting the tarso-metatarsal joints, neuropathic joint changes in diabetes have been described in the ankle and knee (Shore 1947, Spear 1947). In 1958 Jacobs, reporting three new patients, estimated that only forty-five patients with this condition had been recorded in the world literature up to 1956. We are reporting here two further cases.

CASE REPORTS

Case 1—A man aged thirty-five, a post-office engineer, developed diabetes while in the Army in 1945. Although he had unstable periods with intercurrent infection, for many years his daily requirements had been 40 units of protamin zinc insulin and 20 units of soluble insulin, on a diet containing 160 grammes of carbohydrate. In 1957 the diabetes became unstable

FIG. 1

FIG. 2

FIG. 3

Case 1. Figure 1—The swelling of the right foot was diffuse and painless. Figure 2—Radiograph of the right foot taken at the same time as the clinical photograph. Figure 3—The radiographic changes after three months' treatment.

and on three occasions he was admitted to hospital with threatened coma. Insulin zinc suspension was tried but found to be of no help.

In April 1958 he injured the little toe of the right foot with a spade, with bruising but no pain. A few days later the skin broke, the toe became infected and he was admitted to hospital. A radiograph showed only subluxation of the middle phalanx of the little toe. Gangrene occurred and part of the toe was lost, but healing was satisfactory. The foot retained a good circulation and the blood pressure was 155/95 millimetres of mercury. He

was in hospital for two months and in bed for most of the time. Two weeks after his discharge a diffuse painless swelling of the right foot was first noticed (Fig. 1), with no inflammatory reaction; the radiographs showed destruction of the tarso-metatarsal joints of the right foot and new bone formation (Fig. 2). The knee and ankle jerks were absent on both sides and the plantar responses flexor. Vibration sense was absent on the right side below the knee, and diminished on the left. Position sense was normal but there was a patchy loss of pain and touch sensation. The fundi showed moderately advanced diabetic retinitis. The urine contained no albumin but usually one-quarter to one-half per cent of sugar; if the latter was not present he tended to have hypoglycaemic symptoms. The Wassermann reaction and Kahn test were negative.

Remaining fairly well controlled on 16 units of protamin zinc insulin and 44 units of soluble insulin, the patient has shown a gradual clinical and radiological improvement in the foot (Fig. 3).

Case 2—A married woman, aged seventy-five, and known to have had diabetes for nine years, was usually well controlled on a low carbohydrate diet only. She was admitted to hospital in 1958 for swelling of the left leg below the knee which had started in the foot over seven weeks before. A year previously she had had a feeling as if the arch of the foot had given away. The ankle and knee reflexes were normal; the plantar responses were flexor. The vibration sense was absent, and pain and touch were diminished from below the knee and position sense was normal. She had varicose veins in the left thigh. Radiographs showed some erosion of the tarso-metatarsal joints with loss of joint space in the left foot and extensive periosteal new bone formation around the shaft of the first metatarsal (Fig. 4). A biopsy of bone showed an irregular absorption and formation of bone and disorganisation of the articular cartilage, but no inflammatory changes were seen. The fasting blood sugar was 198 milligrams per cent. The Wassermann reaction and Kahn test were negative; the blood uric acid, blood urea, serum calcium and alkaline phosphatase were normal. The diabetes was controlled on a diet containing 160 grammes of carbohydrate and Tolbutamide 1 tablet twice daily, with a steady clinical and radiographic improvement of the foot.

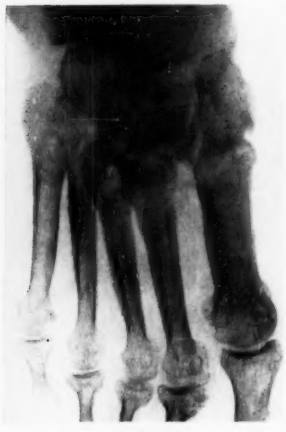


FIG. 4
Case 2—The left foot at time of diagnosis.

DISCUSSION

These two patients are reported to draw attention to this rare complication of diabetes. Osteomyelitis, tuberculosis and tumour must be considered in the differential diagnosis; in 1951 Lister and Maudsley described a patient with this complication which was treated for some time as tuberculosis, and a biopsy was done before the correct diagnosis was made.

We believe that the prognosis of neuropathic changes in joints is better in patients with diabetes than in those with tabes dorsalis or syringomyelia, and that the earlier the diagnosis is made and the appropriate treatment given the better the chance of improvement in the affected joints.

Eloesser in 1917 showed that repeated small injuries are necessary in a desensitised joint to produce neuropathic changes. In the patients here reported there was definite diabetic neuropathy as shown by absent knee and ankle jerks, loss of vibration sense and diminution of pain and touch sensation.

Foster and Bassett (1947) placed the neurological lesion as "predominantly extramedullary, with the major involvement in the posterior root ganglia and the sensory components of the

peripheral nerves with relative sparing of the ventral roots and motor fibres of the peripheral nerves."

All reports agree that the small joints of the feet, especially the tarso-metatarsal and metatarso-phalangeal, are most commonly involved, and occasionally the ankle or knee. Perhaps this is because slight often-repeated injury is more likely to affect the small joints of the foot, with their comparatively weak capsule and ligaments, than the larger and stronger joints of the leg. Vascular insufficiency appears to play no part.

The first sign is a painless swelling of the foot, without heat or redness. This occurred in all seventeen patients recorded by Bailey and Root (1947). The radiographic changes include erosion at the joint surfaces, destruction of the joint spaces and extra-articular new bone formation. It is interesting that these joint changes were reversed in Case 1, with some improvement; Jacobs (1958) reported a similar finding in one of his patients. It is possible that in diabetes the neuropathy is reversible if nerve changes have not progressed too far. The most recent radiographs of our patients (May 1959) show improvement continuing, with absorption of extra-articular calcification and improved definition of the joint surfaces.

Treatment consists in stabilisation of the diabetes, physiotherapy—particularly faradism—and supporting bandages or stockings and surgical footwear.

SUMMARY

Two cases of neuropathic joints in diabetes mellitus are described. The condition, though rare, must be kept in mind in diabetes with neuropathy. With proper control of the diabetes and supportive measures to the joints the prognosis is relatively good.

We should like to thank Dr W. A. Jaques, Senior Consultant Physician, and Mr E. P. Waters, Consultant Orthopaedic Surgeon, for allowing us to publish these cases, and the Photographic Department of the Friarage Hospital, Northallerton, for the photographs.

REFERENCES

BAILEY, C. C., and ROOT, H. F. (1947): Neuropathic Foot Lesions in Diabetes Mellitus. *New England Journal of Medicine*, **236**, 397.

ELOESSER, L. (1917): On the Nature of Neuropathic Affections of the Joints. *Annals of Surgery*, **66**, 201.

FOSTER, D. B., and BASSETT, R. C. (1947): Neurogenic Arthropathy (Charcot Joint) Associated with Diabetic Neuropathy: Report of Two Cases. *Archives of Neurology and Psychiatry*, **57**, 173.

JACOBS, J. E. (1958): Observations of Neuropathic (Charcot) Joints Occurring in Diabetes Mellitus. *Journal of Bone and Joint Surgery*, **40–A**, 1,043.

JORDAN, W. R. (1936): Neuritic Manifestations in Diabetes Mellitus. *Archives of Internal Medicine*, **57**, 307.

LISTER, J., and MAUDSLEY, R. H. (1951): Charcot Joints in Diabetic Neuropathy. *Lancet*, **ii**, 1,110.

MARTIN, M. M. (1953): Diabetic Neuropathy. A Clinical Study of 150 Cases. *Brain*, **76**, 594.

SHORE, T. H. G. (1947): Diabetic Neuropathy. Letter to the Editor. *Lancet*, **ii**, 738.

SPEAR, G. E. (1947): Diabetic Arthropathy. Letter to the Editor. *Lancet*, **ii**, 963.

CARPO-METACARPAL DISLOCATION

Report of Four Cases

EDMUND SHEPHARD, MAIDSTONE, and D. J. SOLOMON, TUNBRIDGE WELLS, ENGLAND

Carpo-metacarpal dislocation other than of the thumb alone is uncommon. Waugh and Yancey (1948) collected eighty-three published cases, and added two of their own. Picchio (1954) listed 100 instances.

Complete carpo-metacarpal dislocation—that is, of all five metacarpals of one hand—is rare. Waugh and Yancey (1948) drew attention to five recorded cases, those of Rivington (1873), Poulet (1884), McLean (1922), Michaelsshon cited by Mittler (1937), and Triksen cited by Binotto (1940).

Aulong and Girard (1951) reported a further case of complete carpo-metacarpal dislocation with fracture of the base of the fifth metacarpal bone. They were unwilling to accept Poulet's and Triksen's cases because the evidence was based only on plaster casts. Picchio (1954) reported no additional case of complete carpo-metacarpal dislocation. In Trojan's (1952) patient the second to fifth metacarpals were dislocated dorsally, but the trapezium was displaced with the first metacarpal, the first carpo-metacarpal joint not being disturbed.

Waugh and Yancey recorded thirty-one cases of dislocation of the four medial carpo-metacarpal joints together, and eleven cases, including two of their own, of combined dislocation of the fourth and fifth metacarpal bases.

Four cases are here presented: one of dislocation of all five carpo-metacarpal joints of one hand; one of dislocation of the four medial carpo-metacarpal joints with subluxation of the corresponding thumb joint; one of dislocation of the four medial carpo-metacarpal joints; and one of dislocation of the fourth and fifth carpo-metacarpal joints.

CASE REPORTS

Case 1—A woman of fifty-six was struck by a car, sustaining severe multiple injuries to trunk and limbs. The left hand showed dorsal dislocation of all five metacarpals on the carpus (Fig. 1), and a fracture of the second metacarpal neck, over the dorsum of which was a small laceration. At operation undertaken for major injuries, reduction of the left carpo-metacarpal dislocation was attempted without success. A further attempt, just before the patient left the theatre, succeeded at once (Fig. 2), perhaps because the prominent second metacarpal base happened to enter its mortise. The patient died twenty-four hours after the accident.

Necropsy findings—Examination of the left hand at necropsy showed that the dorsal capsule of the carpo-metacarpal joints was intact except for a small longitudinal split. Dislocation was easily reproduced by forward pressure applied to the metacarpal heads. Then the metacarpal bases overlapped the carpus by one inch. From the dorsum of the carpus, and from the front of the metacarpal bases the soft tissues were found stripped for one inch. After redislocation, reduction was impossible even by leverage, until the extensor tendons were divided, when it became easy.

Case 2—A man of twenty-four, a tree feller, rode his motor cycle head-on into the side of a shooting brake. On his admission to the Kent and Sussex Hospital there was a large haematoma on the dorsum of the left hand and distal half of the forearm. Radiographs (Fig. 3) showed dorsal dislocation of the four medial metacarpal bases, and subluxation of the first metacarpal base. In addition, the hamate, capitate, trapezium, scaphoid tuberosity and fifth metacarpal base showed fracture.

FIG. 1
Case 1—Left hand. Dorsal dislocation of all five carpo-metacarpal joints, and fracture of second metacarpal neck.

FIG. 2
Case 1—Radiograph after reduction.

To facilitate manipulative reduction under anaesthesia, blood and clots amounting to even ounces (200 millilitres) were evacuated through a short incision on the dorsum of the hand. Reduction was then obtained easily by traction on the thumb and four fingers, both thumbs of the operator applying pressure on the metacarpal bases. The wound was drained and an anterior plaster splint applied with the wrist extended.

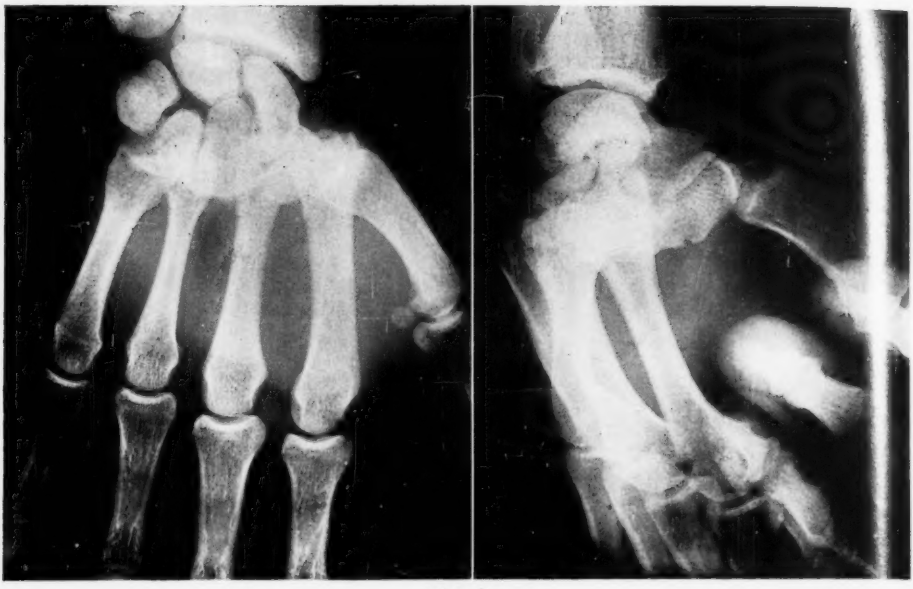


FIG. 3

Case 2—Left hand. Dorsal dislocation of the four medial carpo-metacarpal joints and subluxation of the corresponding thumb joint with fractures of hamate, capitate, trapezium, scaphoid tuberosity and fifth metacarpal base.

FIG. 4

Case 2—Radiograph after reduction.

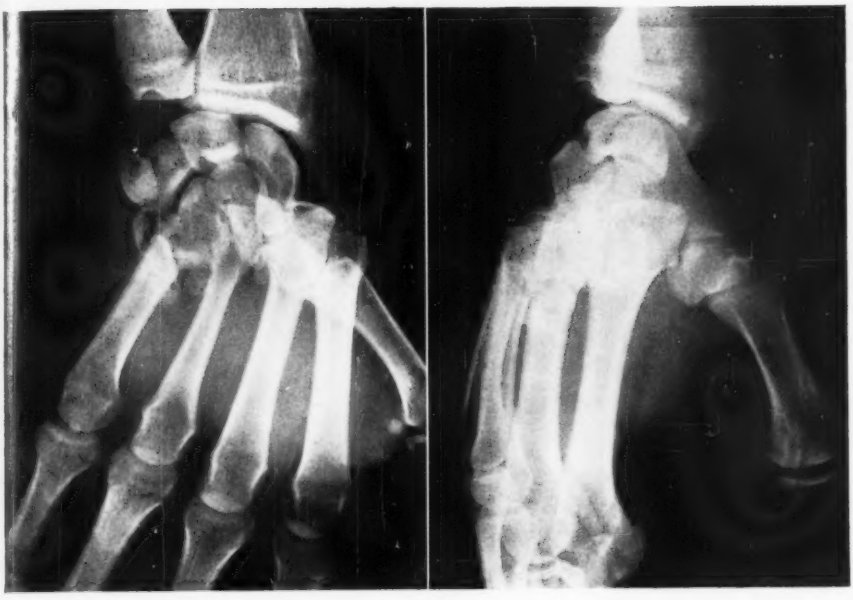


FIG. 5
Case 3—Right hand. Dorsal dislocation of the four medial carpo-metacarpal joints.

FIG. 6
Case 3—Radiograph after reduction.

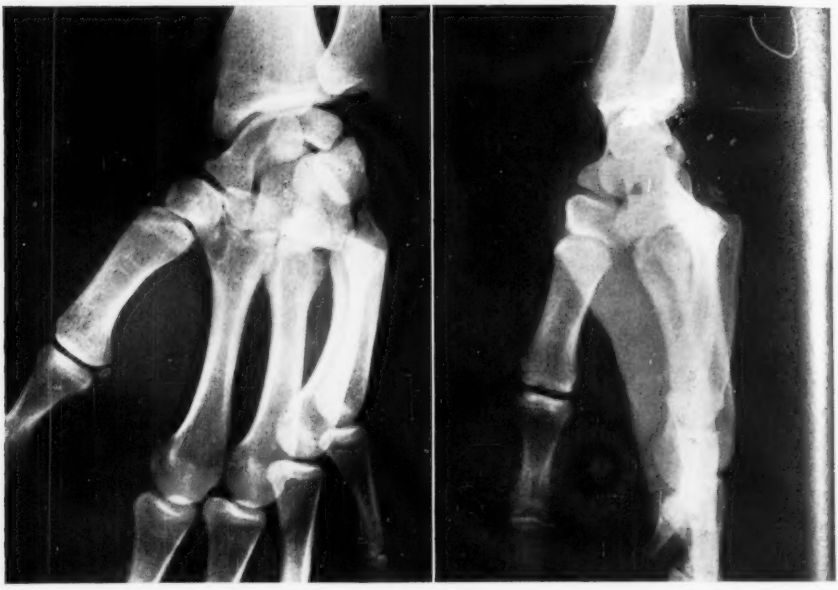


FIG. 7
Case 4—Left hand. Dorsal dislocation of fourth and fifth carpo-metacarpal joints, with flake fracture of hamate bone.

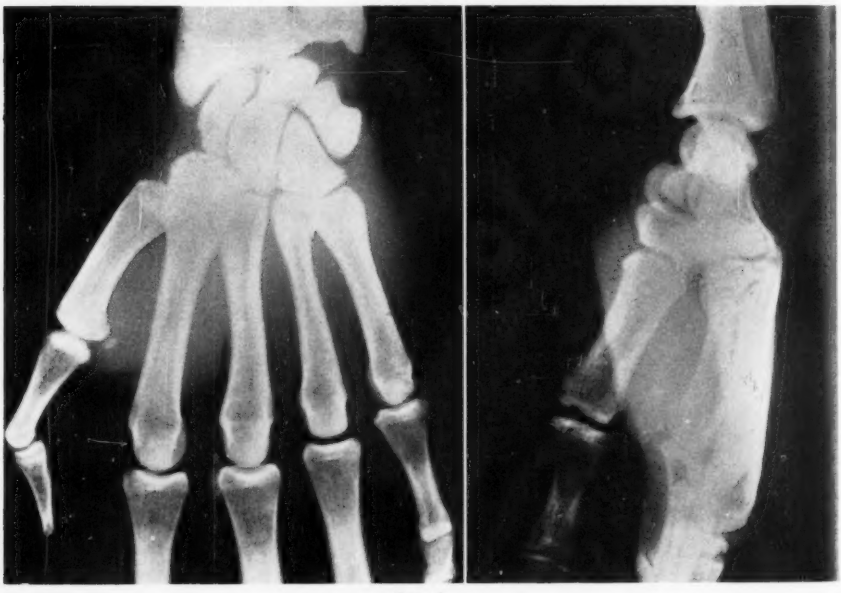


FIG. 8
Case 4—Radiograph after reduction.

Radiographs (Fig. 4) showed reduction, with slight persistent dorsal displacement of the fourth and fifth metacarpal bases, associated with comminution of the hamate.

The drain was removed after forty-eight hours; thereafter a plaster case was applied and maintained for six weeks. Three months after the injury function was excellent and the grip strong. The fourth and fifth metacarpal bases were slightly prominent on the dorsum of the hand. The patient is now working as a self-employed contractor.

Case 3—A man of eighteen, a fitter-turner apprentice, was admitted to the West Kent General Hospital after a motor-cycle accident with fractures of the right thigh and leg, and dorsal dislocation of the four right medial carpo-metacarpal joints (Fig. 5). At the initial operation the hand injury was disregarded, but two days later the carpo-metacarpal dislocation was reduced easily under anaesthesia, and a plaster was applied and maintained for five weeks (Fig. 6). Full movement and function were regained, with slight prominence on the dorsum of the hand.

Return to work has been delayed by the lower limb injuries.

Case 4—A youth of seventeen, of no occupation, attended the Kent and Sussex Hospital with a painful left hand after a motor-cycle accident. He was unable to describe the mechanism of the injury. Radiographs confirmed the clinical diagnosis of dorsal dislocation of the fourth and fifth metacarpal bases, and showed a flake fracture of the hamate bone (Fig. 7). Reduction under anaesthesia was easily gained by traction and local pressure (Fig. 8). A plaster case was applied with the wrist extended, and maintained for six weeks. Function is normal. The patient has enrolled as an art student.

COMMENT

In Cases 2, 3 and 4 the patients were motor cyclists, and the dislocation might be attributed to impact of the handle bar on the metacarpal bases in the palm. Aulong and Girard (1951) so explained their complete dislocation. Pressure by a car steering wheel has also been held responsible.

In Case 1 another mechanism must be adduced. Perhaps the limb was flung outwards until checked by the dorsum of the metacarpal heads striking some object. If the muscles were tensed the momentum of the limb could have been transmitted to the metacarpal bases by abductor pollicis longus, extensores carpi radiales and extensor carpi ulnaris. The blow on the dorsum of the metacarpal heads together with the pull of these extensors may have caused the dislocation.

SUMMARY

Four cases of carpo-metacarpal dislocation are described. In one case all five metacarpals of one hand were involved.

We wish to thank Mr W. Harvey Gervis for permission to report Cases 2 and 4, and for encouragement in the preparation of this paper.

REFERENCES

AULONG, J., and GIRARD, J. (1951): Les luxations carpo-métacarpiennes. *Revue de Chirurgie Orthopédique*, **37**, 439.

BINOTTO, A. (1940): Lussazione volare carpo-metacarpica completa. *Minerva Medica*, **31**, 472.

MCLEAN, E. H. (1922): Carpometacarpal Dislocation. *Journal of the American Medical Association*, **79**, 299.

MITTLER, L. (1937): Sul meccanismo di produzione di una lussazione antero interna del IV e V metacarpo. *Rivista di Chirurgia*, **3**, 487.

PUCHIO, A. (1954): Sulle lussazioni carpo-metacarpiche. *Minerva Chirurgica*, **9**, 43.

POULET (1884): Sub-luxation des cinq métacarpiens sur le carpe, réduction après un mois. *Bulletins et Mémoires de la Société de Chirurgie de Paris*, **10**, 902.

RUSHINGTON (1873): Compound Dislocation of All the Metacarpal Bones of the Right Hand; Operation; Recovery with a Serviceable Hand. *Lancet*, **i**, 270.

TROJAN, E. (1952): Ein Beitrag zu einer seltenen offenen divergierenden Carpo-Metacarpal- und Intercarpalverrenkung der rechten Hand. *Monatsschrift für Unfallheilkunde*, **55**, 65.

WAUGH, R. L., and YANCEY, A. G. (1948): Carpometacarpal Dislocations. *Journal of Bone and Joint Surgery*, **30-A**, 397.

THE TREATMENT OF FRACTURES OF THE MEDIAL EPICONDYLE OF THE HUMERUS

J. N. WILSON, LONDON, ENGLAND

From the Institute of Orthopaedics, Royal National Orthopaedic Hospital, London and Stanmore

Fractures of the medial epicondyle of the humerus, which usually occur in children, are caused by a valgus strain at the elbow joint and the injury is sometimes accompanied by lateral dislocation of the forearm. Permanent disability is rare, but progress is often slow, and, unless a careful follow-up is carried out, a false impression about the final result may be gained. This study was made to find out the influence of different forms of treatment upon the speed of recovery.

MATERIAL

The details of fifty-nine fractures from two hospitals were used in the investigation. The age at the time of the injury varied from five to seventeen years, with the greatest incidence between nine and twelve. All the fractures seemed to have been produced by an avulsion strain, and just over one-third were complicated by a lateral dislocation of the elbow, among which were two examples of displacement of the epicondylar fragment into the joint.

Thus the injuries could be divided into two main types, with thirty-five patients having a simple avulsion and twenty-four patients having a fracture-dislocation. This seemed more realistic than segregating them into intra-articular and extra-articular lesions (Hasner and Husby 1951). It was possible to review personally only twenty-four patients. The duration of observation varied from one to twelve years, with an average of four years.

RESULTS OF TREATMENT

Most of the patients were treated conservatively by immobilisation with the elbow at a right angle, either in plaster or in a collar-and-cuff sling. The fixation was maintained for three weeks on the average; thirty-four patients with avulsion fractures and nineteen with fracture-dislocations were treated in this way. Six patients were treated by open reduction—five with dislocations and one with an avulsion fracture only. In two of these the epicondylar fragment was held in place by a catgut suture; in the others, by a Pidcock pin.

Union—All but one of the displaced avulsion fractures treated conservatively in which follow-up radiographs could be obtained failed to unite. Figures 1 and 2 show a typical result. The remaining one was unusual because the fragment united in the displaced position without the help of surgery (Figs. 3 and 4). All the fractures complicated by dislocation and treated conservatively went on to non-union, except in one patient in whom reduction was very accurate. Open reduction with fixation by suture led to non-union in both. Pin fixation successfully secured bony union in all four patients (Figs. 5 and 6).

Movement—Eight patients had permanent limitation of extension by 5 to 10 degrees. They were equally divided between those with dislocations and those with avulsion injuries. Ten patients took three to six months to obtain full extension, and eight others took six to twelve months.

Pain—Three patients complained of pain on straining the elbow, or when the inner side was knocked. Only one reported any feeling of instability, and this was very slight.

Deformity—A slight cubitus valgus deformity was found in a few patients. Being more apparent than real, it was probably due to prominence of the medial epicondylar fragment. In no patient was there an ugly deformity, even when the fragment was unduly large (Fig. 7).

FIG. 1

FIG. 2

A simple avulsion fracture of the medial epicondyle with distal displacement of the fragment (Fig. 1), and the same elbow four years later showing non-union (Fig. 2).

FIG. 3

FIG. 4

An avulsion fracture of the medial epicondyle (Fig. 3) which was treated conservatively. Two years later it was united in the displaced position (Fig. 4).

FIG. 5

FIG. 6

Pidcock pin has been used to fix a fractured medial epicondyle (Fig. 5). Two years later it has united (Fig. 6). The pin was removed because it was beginning to come out.

Ulnar nerve lesions—There were ulnar nerve symptoms in three patients with a fracture complicating dislocation. This took the form of paraesthesia in the ulnar distribution of the hand. There were no motor signs. In one—in whom the fragment was displaced into the joint—the symptoms still affected the little finger a year later, despite transposition of the nerve.

FIG. 7

A radiograph of a large medial epicondylar fragment which was ununited but gave no cosmetic deformity.

FIG. 8

A radiograph showing the position of the avulsed medial epicondyle immediately after injury in Figure 8, and, in Figure 9, the position two months later when full extension had been obtained, with the fragment lying at the level of the joint.

FIG. 9

DISCUSSION

It is clear from these results that, irrespective of treatment, the final disability from this fracture is slight; yet there is often controversy over the value of open reduction. This must surely be because too much importance is attached to obtaining union of the fragment (Blount, Schulz and Cassidey 1951). Higgs (1936) advised open reduction in displaced fractures because he feared that late ulnar nerve symptoms might result unless perfect reduction and union were obtained. Patrick (1946) on the other hand was unable to find a single example of late ulnar neuritis after uncomplicated fracture of the medial epicondyle. This review has shown that no disability has resulted from the non-union itself, and it seems likely that the only indication for operative treatment is inclusion of the epicondylar fragment within the joint. Patrick (1946) described a method of reduction of the included fragment by applying faradism to the flexor muscles of the forearm. Even though this may be possible, exploration seems the wiser course because of possible damage to the ulnar nerve (Kessel 1953).

Since union of the fracture is of very little importance (Roberts 1934), fixation after an open reduction probably does not matter very much. Stitching the fragment into place will not secure union, and the only chance of success is to use a pin or a screw. Excision of the fragment (McDonnell and Wilson 1948) is not recommended, because the displaced epicondyle is still important in maintaining the contour of the inner side of the elbow. Treatment of the uncomplicated fracture should always be conservative, and consist of immobilisation in plaster for two to three weeks to allow soft-tissue repair to take place on the inner side of the joint. The time-honoured position of the elbow, with the forearm flexed to a right angle, is unsuitable for the treatment of this fracture. It will be observed from the results in this series that the only movement lost was extension, full restoration of which was often very slow. The progress was probably retarded by local injury to the flexor muscles at their origin, combined with a feeling of insecurity at the pseudarthrosis. This delay is also seen sometimes in fractures of the lateral condyle of the humerus with delayed union. Only

when the medial epicondyle has united, or has moved down to the level of the joint line, is full movement regained (Figs. 8 and 9). If we are to accept the principle of conservative treatment, with its inevitable non-union, then it would seem rational to immobilise the elbow in a much more extended position; the avulsed fragment would then be pulled down much closer to its final resting place. Full extension is not recommended, because of the difficulty in mobilising the joint from this position. It is suggested that immobilisation should be at about 60 degrees below the right angle.

SUMMARY

1. Fifty-nine patients with fractures of the medial epicondyle of the humerus have been reviewed, of whom more than one-third also had a dislocation of the elbow.

2. The final disability has been shown to be very slight. Non-union occurs very often with conservative treatment, but gives no disability. Union can be obtained by fixation with a Pidcock pin.

3. Operative treatment is advised only when the fragment is included in the joint. It is suggested that the best position of the elbow in patients treated conservatively is about 60 degrees below the right angle.

I am grateful to Mr Dillwyn Evans and the staff of the Orthopaedic Department of the Cardiff Royal Infirmary for access to their records. My thanks are also due to Mr R. J. Whitley of the Photographic Department of the Institute of Orthopaedics for the illustrations.

REFERENCES

BLOUNT, W. P., SCHULZ, I., and CASSIDEY, R. H. (1951): Fractures of the Elbow in Children. *Journal of the American Medical Association*, **146**, 699.

HASNER, E., and HUSBY, J. (1951): Fracture of Epicondyle and Condyle of Humerus. *Acta Chirurgica Scandinavica*, **101**, 195.

HIGGS, S. (1936): Fractures of the Internal Epicondyle of the Humerus. *British Medical Journal*, **ii**, 666.

KESSEL, A. W. Lipmann (1953): Fractures Around the Elbow Joint in Children. *Postgraduate Medical Journal*, **29**, 33.

MCDONNELL, D. P., and WILSON, J. C. (1948): Fractures of the Lower End of the Humerus in Children. *Journal of Bone and Joint Surgery*, **30–A**, 347.

PATRICK, J. (1946): Fracture of the Medial Epicondyle with Displacement into the Elbow Joint. *Journal of Bone and Joint Surgery*, **28**, 143.

ROBERTS, N. W. (1934): Displacement of the Internal Epicondyle into the Elbow-Joint. *Lancet*, **ii**, 78.

LATERAL DISLOCATION OF THE RADIO-HUMERAL JOINT WITH GREENSTICK FRACTURE OF THE UPPER END OF THE ULNA

F. HOWARD BEDDOW and P. H. CORKERY, LIVERPOOL, ENGLAND

From the Alder Hey Children's Hospital, Liverpool

In 1957 Hume described anterior dislocation of the head of the radius associated with undisplaced fracture of the olecranon in children. Of the three cases that he described and illustrated it is our opinion that, whereas Cases 2 and 3 were classical examples of the injury, Case 1 is of an entirely different entity, namely a lateral dislocation of the radio-humeral joint with greenstick fracture of the upper end of the ulna. We have recently encountered two patients with the same injury.

FIG. 1
Case 1—Lateral dislocation of the radio-humeral joint with greenstick fracture of the upper end of the ulna. Before reduction.

FIG. 2
Case 1—After reduction.

FIG. 3
Case 1—Five months later.

CASE REPORTS

Case 1—A boy aged six years sustained an injury to his right elbow in falling from a tricycle. Radiographs showed an oblique greenstick fracture of the upper end of the ulna with lateral dislocation of the radial head (Fig. 1). Reduction was done with ease under general anaesthesia (Fig. 2). The elbow was rested in a collar and cuff sling for four weeks. Normal movement was regained in two further weeks and radiographs at five months were indistinguishable from normal (Fig. 3).

Case 2—A boy aged four years fell from his tricycle and injured his left elbow. Radiographs showed a greenstick fracture of the ulna at the level of the superior radio-ulnar joint, with lateral displacement of the radio-humeral joint (Fig. 4). There was paralysis of the extensor pollicis longus and extensor digitorum communis. Reduction was performed under general anaesthesia (Fig. 5), and the elbow was rested in a collar and cuff sling at 45 degrees for four weeks. Two weeks later there was a full and painless range of elbow movement and a complete

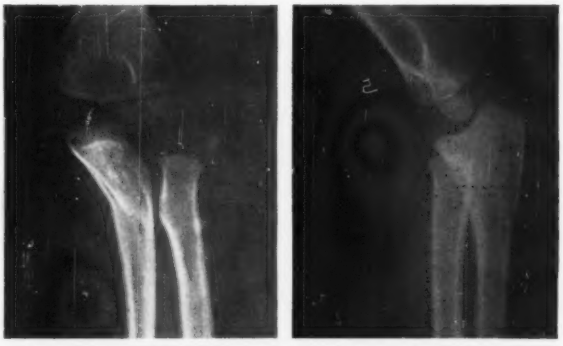


FIG. 4
Case 2—Lateral dislocation of the radio-humeral joint with greenstick fracture of the upper end of the ulna. Before reduction.

FIG. 5
Case 2—After reduction.

FIG. 6
Case 2—Three months later.

recovery from the radial nerve lesion. Three months later radiographs showed appearances indistinguishable from the normal (Fig. 6).

DISCUSSION

In our records are radiographs of two further cases. The injury appears to be consistently an oblique greenstick fracture of the upper end of the ulna at the level of the superior radio-ulnar joint with slight lateral angulation. The lateral view shows the posterior cortex to be split off as a longitudinal "flake." The head of the radius is dislocated laterally at the radio-humeral joint (Figs. 1 and 4).

It is interesting that in Hume's Case 1 and in two of our patients the injury was caused by the child's falling from a cycle.

Reduction was easy under general anaesthesia and consisted simply of correcting the varus deformity by pressure over the head of the radius with the elbow in extension. The elbow was rested at 45 degrees in a collar and cuff sling for four weeks.

One patient had a radial nerve lesion. This was complete at first, but recovery was rapid and complete without any special treatment. It is believed that there was a neurapraxia from traction by the displaced radius on the nerve as it winds through the supinator brevis.

We believe that it is important to draw attention to this lesion, firstly because it may be associated with a radial nerve lesion which (so far as we are aware) has not been described with anterior dislocation, and secondly because immobilisation in plaster-of-Paris appears to be unnecessary.

SUMMARY

1. Two cases of lateral dislocation of the radio-humeral joint with greenstick fracture of the upper end of the ulna are described.
2. One case was complicated by a radial nerve lesion.
3. Treatment was by reduction under general anaesthesia and resting the elbow in a collar and cuff sling. Full recovery was present in six weeks.

We wish to thank Professor B. L. McFarland for help and advice, and Mr F. C. Dwyer and Mr G. L. Shatwell for permission to publish these cases.

REFERENCE

HUME, A. C. (1957): Anterior Dislocation of the Head of the Radius Associated with Undisplaced Fracture of the Olecranon in Children. *Journal of Bone and Joint Surgery*, **39–B**, 508.

TUMOURAL GUMMATOUS YAWS

W. P. COCKSHOTT, IBADAN, NIGERIA

Radiologist, University College Hospital and University College, Ibadan

and

A. G. M. DAVIES, KAMPALA, UGANDA

Radiologist, Mulago Hospital, Kampala

We have seen from time to time patients showing an unusual change in bones which we attribute to a treponematose, probably yaws. As this particular type of bone lesion does not seem to be well recognised and is not described in the standard work of Hackett (1951) on the bone lesions of yaws, two examples of this process are here described, one from Nigeria and one from Uganda.

FIG. 1

FIG. 2

Case 1. Figure 1—Right forearm showing expanding inflammatory process affecting distal third of both forearm bones. Figure 2—Brachial arteriograph showing the avascular nature of the lesion.

Case 1—A Yoruba tailor aged twenty-seven attended University College Hospital, Ibadan, giving a history of two years' painless swelling of the lower forearm. The swelling was firm, of normal temperature and not pulsatile. Movements of the overlying tendons were

unimpeded and the skin was mobile over the tumour. The tumour had at first increased rapidly in size but growth had been stationary for some time. A radiograph (Fig. 1) disclosed an unusual productive and destructive lesion affecting the distal third of the radius and ulna.

On the medial side of the ulna there was considerable organised fusiform accretion of periosteal new bone which was also present at the proximal end of the radial lesion. A fairly abrupt transition occurred between this zone and an area where there was no remnant of the original bony structure. The normal bony tissue had been replaced by a soft-tissue mass which contained many small lace-like fragments of bone giving rise to a bizarre mottled appearance.

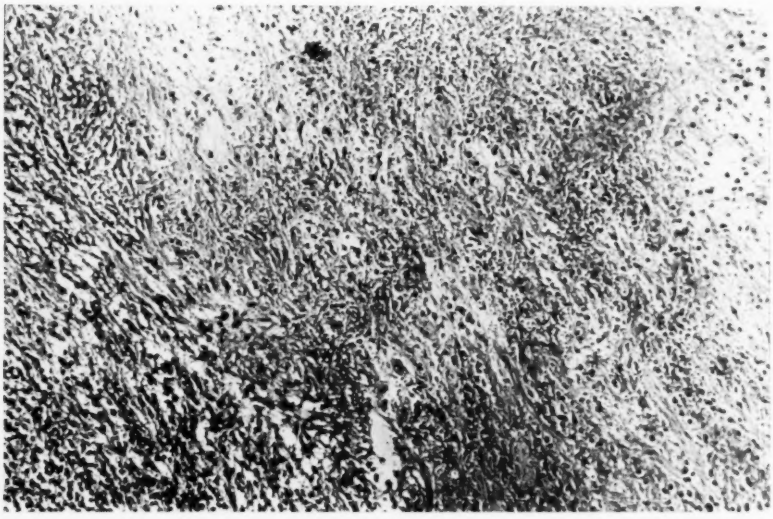


FIG. 3
Case 1—Edge of structureless necrotic area showing round-cell infiltration and absence of capillaries. (×120.)

Brachial arteriography showed the lesion to be displacing normal vessels by reason of its mass, but there were no new vessels and indeed the area seemed to be relatively avascular (Fig. 2). The Kahn reaction was strongly positive. A treponemal immobilisation test could not be performed. Biopsy revealed a relatively structureless material with areas of necrosis (Fig. 3). Within the almost acellular eosinophilic matrix small irregular bony trabeculae were present. A few multinucleated giant cells were evident and occasional lymphocytes. The tissue was strikingly avascular.

Case 2—A Lango tribesman aged twenty-five attended Mulago Hospital, Kampala, complaining of weakness of his hand and swelling of his left forearm. He gave a history of having had a generalised yaws eruption in childhood and also of vague bone pains two years previously. On examination there was a fusiform swelling of the forearm which was hard and tender on pressure. Some wasting of the small muscles of the hands was noted, and there was considerable loss of power of the grip.

Radiography disclosed marked fusiform expansion of the distal two-thirds of the radius and to a lesser extent of the ulna (Fig. 4). This was due to exuberant periosteal new bone formation combined with a lack of structure of the medulla. The same type of mottled

appearance noted in the last case was again seen. Extension of periosteal new bone in the line of the interosseous ligament was observed in the proximal part of the lesion.

Biopsy revealed a fibrous structureless matrix containing a few inflammatory cells. The bony trabeculae showed little activity. Some perivascular cuffing and thickening of small arteries was noted. This was considered to represent tertiary yaws osteitis.

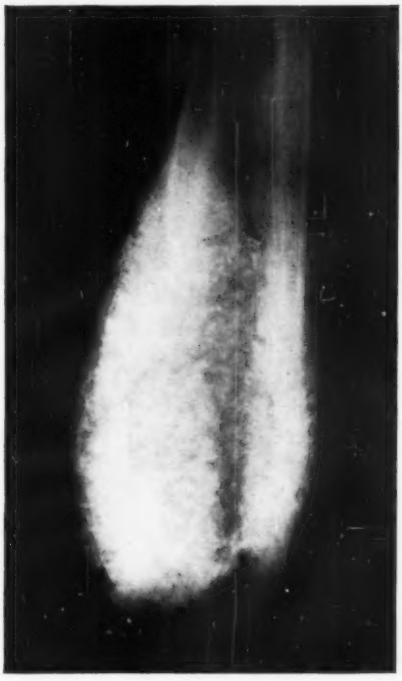


FIG. 4
Case 2—Massive patchy new bone formation affecting both forearm bones. Strands of new bone extend in the line of the interosseous ligament.

DISCUSSION

Several examples of this type of process have been seen in the last two years in Ibadan. They have all been in young men with positive serological tests and no obtainable history of syphilis. The dominant lesion has always been in the upper limbs and the tumour mass appears to have a predilection for the end of a bone. One patient had multiple lesions including a periosteal dactylitis and a subcortical gumma at the upper end of the tibia in addition to a gummatous lesion of the lower end of the humerus.

The radiographic response to penicillin has been negligible in these patients, though none has reported for follow-up later than six months from the beginning of treatment.

The distinction between yaws, syphilis and endemic syphilis is not possible radiologically, and even with refinements in serological investigation, such as the treponemal immobilisation test, no absolute classifying criteria are available. A careful history and a knowledge of the local prevalence of these allied conditions is the best guide.

We consider this type of lesion to be a manifestation of gummatous tertiary yaws.

REFERENCE

HACKETT, C. J. (1951): *Bone Lesions of Yaws in Uganda.* Oxford: Blackwell Scientific Publications.

JAVELIN THROWER'S ELBOW

J. E. MILLER, MONTREAL, CANADA

From The London Hospital, London, England

In February 1959 a woman of eighteen sought advice in the Orthopaedic and Acci ent Department of The London Hospital because of pain in the right elbow. She was or of Great Britain's leading javelin throwers and a promising contender for a position ir the Olympic team.

She first experienced this pain in 1956, a year after she had become interested in ja elin throwing. The symptoms, initially slight, were situated on the medial and extensor a ect of the elbow, occurring at the moment of throwing and then resolving within a few mom nts; or alternatively, appearing as a dull, diffuse ache and stiffness on the day after throv ng. The pain progressively increased and in 1957 it was a constant accompaniment of her efl rts. She attended another hospital and received a course of five injections of hydrocortisone nto the tender medial side of the elbow, with considerable relief.

FIG. 1

Right elbow showing irregularity at the tip of the olecranon.

In the autumn of 1958 the pain recurred and was so severe that it prevented her from throwing. With a few isolated throws, at the instant of full extension of the elbow she experienced, in addition to elbow pain, a transient paraesthesia in the ulnar distribution of the hand.

The elbow was normal on clinical examination but radiographs (Fig. 1) showed irregularity at the tip of the olecranon.

In April 1959 the ulnar nerve was explored at the elbow. It was found to be slightly constricted at the point where it passed between the heads of flexor carpi ulnaris, and it was

therefore transposed anteriorly. The olecranon was then exposed and was found to be fractured about 0·8 centimetre from the tip (Fig. 2). The small fragment was removed and the triceps reconstituted.

Recovery was rapid. Two months after operation she began to train and six months later had surpassed her previous best throw.

FIG. 2
Photograph at operation showing a transverse fracture of the tip of the olecranon.

DISCUSSION

It was the impression of the writer that elbow pain in javelin throwers is an unusual complaint. Extensive inquiry, particularly from trainers, coaches and athletes, soon established that it is, in fact, common. It is experienced at some time by almost every competitor, varying from slight discomfort to symptoms severe enough to prevent throwing. In spite of its frequent occurrence and its disabling and discouraging effect, the condition appears to have stimulated little interest. The literature is practically devoid of reports.

No mention of the syndrome has been found in the dissertations of the ancients, even though it might well have been endemic at a time when wars were fought with hurled missiles. The English medical literature of the past forty years does not contain a single report of pain in the elbow in javelin throwers. Most text-books on athletic injuries fail to mention the condition and not one discusses it in detail. In the German and Scandinavian literature Heiss (1934) and Waris (1946) published radiological surveys of the changes in a number of elbows which were symptomless at the time of the study.

A similar condition commonly occurs in the elbows of professional baseball pitchers (Bennett 1941, 1947) and may be severe enough to terminate their careers.

Clinical features—" Javelin elbow " or " javelin thrower's elbow " is a condition characterised by pain in the elbow during or after throwing. It is most commonly seen in the unskilled and untrained enthusiast, but can develop in any calibre of javelin thrower. The cause is not completely understood but it appears to be related to an imperfect throwing technique. The onset is usually insidious, in the form of slight discomfort on the inner aspect of the elbow during the throw, or a generalised ache and stiffness after a strenuous session. The pain tends to increase in proportion to the amount and intensity of throwing until eventually it may interfere with the performance of the athlete. Conversely, it tends to resolve with rest.

Examination of the elbow reveals tenderness in the course of the medial ligament, usually over the medial epicondyle and, less commonly, over the ulnar attachment of the ligament. There is a full range of painless movement and resisted effort of the elbow and wrist does not reproduce the symptoms. The signs disappear with a period away from throwing.

Radiographs of the elbow are usually normal. Uncommonly, spur formation or calcification is noted on the medial side of the elbow and rarely, irregularities of the tip of the olecranon are seen.

FIG. 3

FIG. 4

FIG. 5

FIG. 6

The "round arm" method of arm action. Note that the elbow comes around at the level of the shoulder (Figs. 4 and 5) and that medial rotation of the shoulder (Figs. 5 and 6) transmits the force to the javelin.

DISCUSSION

Elbow pain is such a common affliction of javelin throwers that the serious athlete regards it as an unpleasant and often unavoidable accompaniment of the sport and seldom seeks medical advice. Most coaches who guide the enthusiasts and deal with their complaints believe that the condition is the result of an incorrect throwing technique (Lockwood 1960).

The successful running throw requires the highest degree of coordination of limbs and trunk to effect a transfer of momentum to the javelin. In the absence of a smooth train of movements the javelin is delivered by a short quick jerk of the arm which may strain or injure the elbow. In addition to general lack of coordination in the throw, the action of the propelling arm is of the greatest significance in the development of pain.

There are two types of arm motion in general use and these result in two definite types of "javelin elbow." The limited information available in the literature and from personal communications suggests that each type is characterised by distinctive clinical and radiological findings.

The common type usually develops in the novice and uncoached thrower who employs a method known as "round arm" or "side arm" (Figs. 3 to 6). As the action of the arm begins, the elbow swings around in an arc at about the level of the shoulder. With the arm in the abducted position parallel to the ground, and with the elbow flexed to a right angle, medial rotation of the shoulder transmits the required force to the javelin but at the same time exposes the medial ligament of the elbow to a considerable strain. Damage appears to be cumulative rather than the result of a single incident (Baetzner 1936, Heiss 1934). Recurrent minor strains of the ligament result in the common type of "javelin elbow" which is characterised by pain and tenderness at the medial aspect of the joint. The pain is felt before extension of the elbow begins (Lockwood 1960), at the moment when the medial ligament is transmitting the greatest force. It interferes to a variable degree with the performance of the thrower and quickly resolves with rest, to recur with the next session of throwing. The exact nature of the injury is not certain, but appears to be related to the medial ligament of the elbow.

The late sequel of these recurrent strains is new bone formation in the form of spurs or ossicles on the medial side of the joint (Heiss 1934, Bennett 1947). Nothing is known about the degree and duration of re-injury required to produce these changes.

There are no recognised principles in the treatment of this type of "javelin elbow" other than reduction in the intensity of throwing with increased emphasis on the employment of

correct coordination and technique. Severe symptoms may demand complete cessation of throwing for a variable period, during which time general training should continue and can include putting the shot and discus throwing. Established symptoms often resolve when the thrower abandons " round arm " in favour of the correct method (Lockwood 1960). Acute symptoms respond temporarily to injected local anaesthetic, allowing unimpaired throwing for several hours before the disability reappears unchanged (Nicholson 1960). Considerable

FIG. 7

FIG. 8

FIG. 9

FIG. 10

The correct method of arm action. Note that the elbow, remaining flexed, takes up a position well above and in front of the shoulder and close to the head (Figs. 8 and 9). Extension of the elbow (Fig. 10) launches the javelin.

improvement often follows the injection of hydrocortisone into the tender area (Newman 1960). Spur formation and ossicles are the source of symptoms only rarely and removal in these cases affords relief (Bennett 1941).

The second method of throwing, the correct method (Figs. 7 to 10), is somewhat more difficult to learn than the " round arm " technique and may result in " javelin elbow " distinctly different from the common type. The elbow is brought well forward early in the throw so that it reaches a position directly in front of and above the shoulder. Powerful extension of the elbow then transmits the necessary force to the javelin without endangering the medial ligament. This is the method which is taught by most coaches. It is seldom mastered completely by throwers, but the employment of its principles, even incompletely, tends to prevent the development of " javelin elbow " of the medial ligament type.

Rarely, the accomplished javelin thrower using this method may hyperextend the elbow at the end of the throw, impinging and injuring the tip of the olecranon process against the floor of the olecranon fossa. Waris (1946), who examined seventeen outstanding Finnish throwers radiologically, found changes at the tip of the olecranon in ten. These varied in degree from roughness and irregularity to complete detachment. In one there was a fracture of the entire olecranon with malunion. All had suffered pain in the elbow after one particular throw or " mal-throw " which had disabled them for periods from a few weeks to as long as a year and which had recurred periodically.

When surgical excision of the loose tip has been practised it has produced a complete cure (Welply 1960).

There is little doubt that prevention is of great importance. Employing the correct throwing technique removes or minimises the threat of " javelin elbow." It is essential that the novice receive careful and conscientious guidance in his earliest efforts from a competent coach. To withhold instruction until the individual's interest has deepened or until he shows promise is a prelude to the development of " javelin elbow." Bad technique, once established, is difficult to correct and tends to reappear when the athlete is " off his form."

The management of the condition is a matter for close cooperation between the surgeon, the patient and the coach.

The patient described in this report first suffered from the common type of " javelin elbow " due to medial ligament strain, and this responded to injections of hydrocortisone.

The recurrence of severe pain in 1958 came at a time when the patient was making her best throws and probably marked the injury to the tip of the olecranon.

The symptoms related to the ulnar neuritis are unusual and no other similar history has been encountered.

SUMMARY

1. Pain in the elbow in javelin throwers is a common complaint.

2. The commonest type is caused by recurrent strain of the medial ligament. It develops in individuals who employ an incorrect throwing technique. The symptoms are cumulative, increasing with throwing and decreasing and resolving with rest. Treatment consists in improving the throwing technique. Local anaesthetic injected into the tender area produces complete but temporary relief. Hydrocortisone may produce partial or complete relief.

3. A second type of "javelin elbow" occurs in expert throwers and is the result of hyperextension of the elbow at the end of the throw, causing an injury to the tip of the olecranon. The symptoms are the result of a single throw or "mal-throw" and are completely disabling. They resolve with rest but tend to recur. If the tip of the olecranon is fractured excision of the fragment completely relieves the symptoms.

This patient was treated by Mr H. Osmond-Clarke. I am indebted to him for his permission to publish the report and for his help in the preparation of this paper. I should also like to thank Miss P. Burgess, A.R.P.S., of The London Hospital Photographic Department. It is a pleasure to acknowledge the help of Mr H. H. Lockwood, President of the Ilford Athletic Club, for his valuable guidance and illustrations.

REFERENCES

BAETZNER, W. (1936): As quoted by Waris.

BENNETT, G. E. (1941): Shoulder and Elbow Lesions of the Professional Baseball Pitcher. *Journal of the American Medical Association*, **117**, 510.

BENNETT, G. E. (1947): Shoulder and Elbow Lesions Distinctive of Baseball Players. *Annals of Surgery*, **126**, 107.

HEISS, F. (1934): Entstehung von Knochenbildungen am Ellbogengelenk als Folge starker körperlicher Betätigung. *Deutsche Zeitschrift für Chirurgie*, **242**, 342.

LOCKWOOD, H. H. (1960): *Personal communication.*

NEWMAN, P. H. (1960): *Personal communication.*

NICHOLSON, C. B. (1960): *Personal communication.*

WARIS, W. (1946): Elbow Injuries of Javelin-Throwers. *Acta Chirurgica Scandinavica*, **93**, 563.

WELPLY, W. R. (1960): *Personal communication.*

FORCED INVERSION OF ANKLES

D. H. NELSON, FOLKESTONE, ENGLAND

From the Radiological Department, Royal Victoria Hospital, Folkestone

In hospitals outside the main teaching centres it is seldom possible for a radiologist to survey all wet films and order the necessary supplementary views; it is thus necessary to have routine techniques available at the discretion of the technician. The importance of the forced inversion view of ankles has been stressed for many years but in few departments is it routine.

FIG. 1
Method of self-forced inversion.

In this hospital group it is standard practice to take this additional view of all ankles in patients referred from the casualty department when the antero-posterior and lateral radiographs do not show a fracture. With the method illustrated (Fig. 1) it is unlikely that the patient could inflict any further injury, the assistance of a doctor is not needed, and no second person is exposed to radiation. The technique is not original and may sometimes fail to reveal ligamentous damage, but it has proved effective on many occasions when otherwise there might have been a delay of a day or more in diagnosis by a junior resident in charge of a casualty department.

The injury illustrated in Figures 2 and 3 was picked up by this method and might well have been missed by those who only take a forced inversion view when there is pain and tenderness over the lateral malleolus.

FIG. 2

FIG. 3

Figure 2—Radiographs of ankle of girl of thirteen referred from casualty department. No fracture seen on close scrutiny. Figure 3—Routine self-forced inversion. Fracture of medial malleolus clearly visible.

THE BLOOD SUPPLY OF THE FEMORAL NECK AND HEAD IN RELATION TO THE DAMAGING EFFECTS OF NAILS AND SCREWS

ALFREDO BRODETTI,* OXFORD, ENGLAND

Failures in the treatment of fractures of the femoral neck are caused by several factors. Some of these, such as avascular necrosis of the femoral head, cannot be avoided. Other factors, such as inaccurate reduction or imperfect fixation, can be partly controlled. The possibility has long been considered that the nail itself and the disturbance caused by its introduction might damage the blood vessels of the femoral head. Cedermark (1936) suspected this, and later the studies of Trueta and Harrison (1953) provided the anatomical basis for the clinical suspicions of Watson-Jones (1955) and Hulth (1956) that the nail might damage the vessels of the upper part of the head. In the present work an attempt has been made to determine how the nail or screws may damage the vessels of the femoral head and how the danger may be reduced by correct placing of the fixing agent.

METHODS AND MATERIAL

The vascular patterns described by Trueta and Harrison (1953) for the femoral head, and by Judet, Judet, Lagrange and Dunoyer (1955) for the femoral neck, were taken as the anatomical basis for the study.

Sixteen femoral heads and necks from eight cadavera of persons aged from sixty-two to seventy-six years were used. In addition we were able to study the material collected by Professor J. Trueta. A suspension of barium and Berlin blue (Harrison, Schajowicz and Trueta 1953) was injected into the abdominal aorta. The uppermost third of each femur was then removed and all the surrounding soft tissues were dissected away. Each specimen was radiographed. The specimens were fixed in a vice and a nail or a screw was inserted. These were inserted in different positions and sites: namely, varus (Fig. 1), valgus (Fig. 2) and along the centre of the femoral head and neck (Fig. 3). The last site is referred to as the " neutral zone." The whole specimen was fixed in formalin and the nail was removed. Slab sections approximately half a centimetre thick were then cut perpendicular to the axis of the femoral neck. The number of sections obtained in each case ranged from nine to twelve. The sections were decalcified and prepared by Spalteholz's method. Radiographs were then taken on films with a fine-grain emulsion, using a low voltage unit with a beryllium window. All the radiographs of the slab sections were taken with the medial face of the section towards the tube. The orientation of the specimens was thus kept constant so as to avoid confusion between specimens taken from the right or left femora.

Attempts to nail the femora before they had been injected and removed were abandoned because the profuse leakage from the site of exposure and from the bone itself produced unsatisfactory injections.

RESULTS

Anatomical arrangement of vessels—The vessels entering the femoral head through the round ligament were always found to be numerous (Figs. 4 and 7), and so too were the branches originating from them (Figs. 5 and 7). They were seen to supply the medial third of the femoral head.

* Holder of the Nuffield Scholarship in Orthopaedic Surgery from 1958 to 1959.

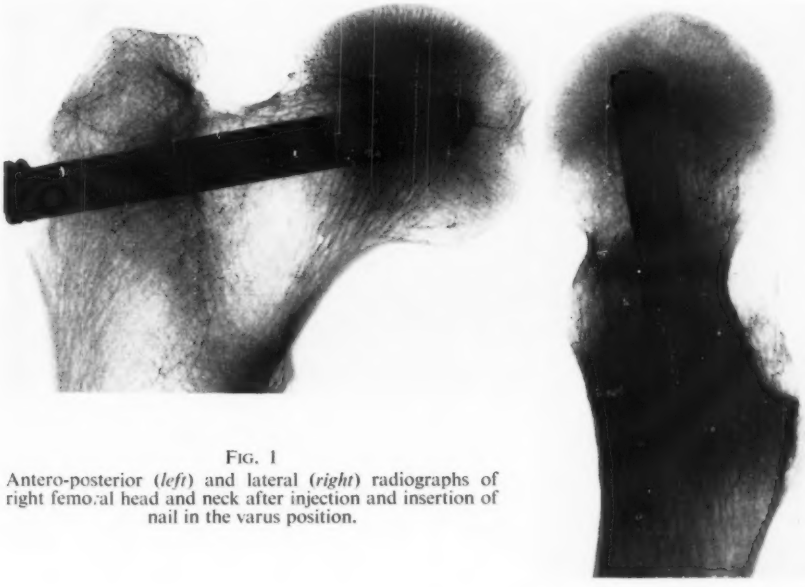


FIG. 1

Antero-posterior (*left*) and lateral (*right*) radiographs of right femoral head and neck after injection and insertion of nail in the varus position.

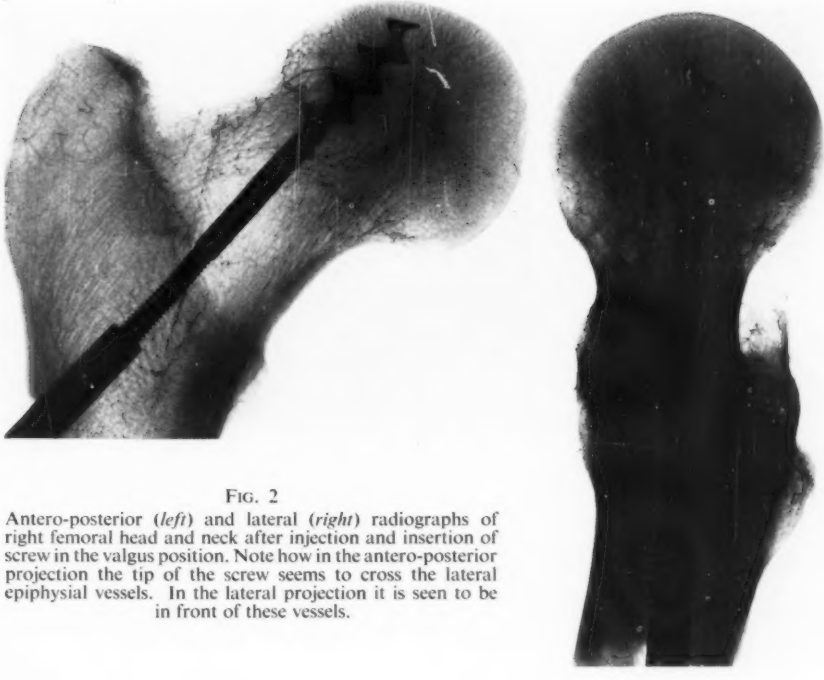


FIG. 2

Antero-posterior (*left*) and lateral (*right*) radiographs of right femoral head and neck after injection and insertion of screw in the valgus position. Note how in the antero-posterior projection the tip of the screw seems to cross the lateral epiphysial vessels. In the lateral projection it is seen to be in front of these vessels.

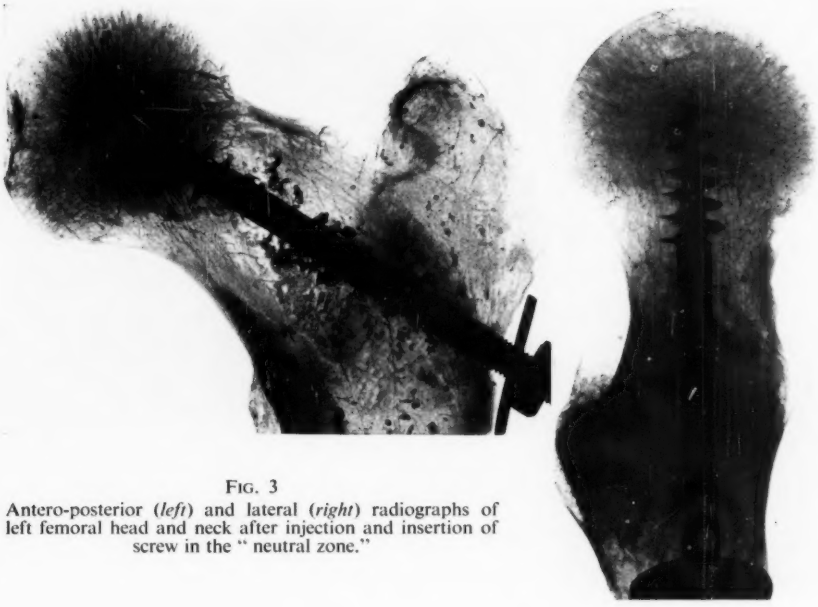


FIG. 3
Antero-posterior (*left*) and lateral (*right*) radiographs of left femoral head and neck after injection and insertion of screw in the " neutral zone."

FIG. 4

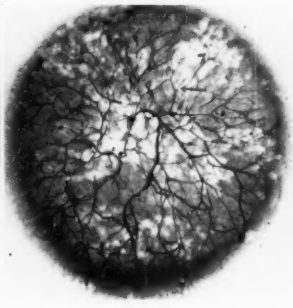


FIG. 5

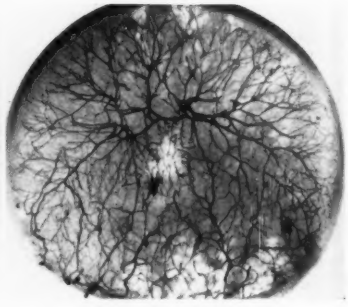


FIG. 6

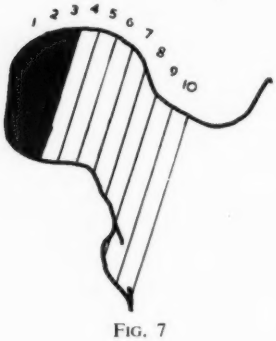


FIG. 7

FIGS. 4 TO 7
Sections through the medial part of a left femoral head after injection (slices 1, 2 and 3 as shown in Figure 7). Figure 4—Slice 1. Showing the vessels entering the head through the round ligament. Figure 5—Slice 2. The rich network of branches arising from the vessels of the round ligament. Figure 6—Slice 3. Showing in the centre of the section the anastomoses between the vessels of the round ligament and the lateral epiphysial vessels.

In all the sections of the femoral head the site and direction of the lateral epiphysial vessels was constant. They anastomosed with the vessels from the round ligament at a variable point which most often was near the junction of the medial and the central thirds of the femoral head (Figs. 6 and 7). These lateral epiphysial vessels cross the central and lateral thirds of the femoral head in its upper and posterior quadrant so that in sections of the right femur (Figs. 8 to 11) they are found on the right side above the centre of the section. In more medial sections they are closer to the centre and in more lateral ones closer to the periphery. In sections of the left femur (Figs. 12 to 15) they lie on the left. This path of the lateral epiphysial vessels was always observed in the sections of the lateral and central zones of the femoral head. They entered the head within an area one centimetre wide located between the cartilage of the head and the cortical bone of the neck. This site of entry is at the upper

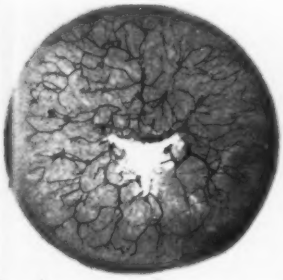


FIG. 8

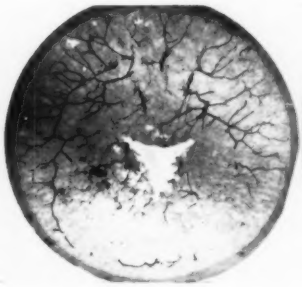


FIG. 9

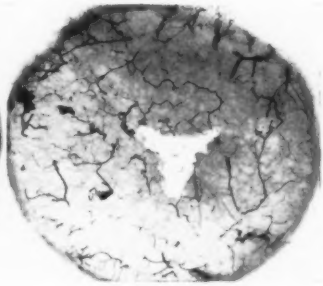


FIG. 10

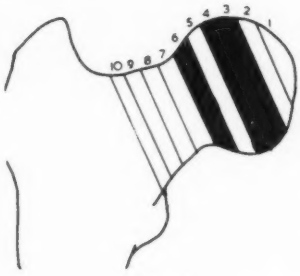


FIG. 11

FIGS. 8 TO 11

Sections through the middle and lateral zones of a right femoral head after injection (slices 3, 4 and 6 as shown in Figure 11). In each section the outline of the track produced by a trifin nail is well shown. Figure 8—Slice 3. The anastomoses between the vessels of the round ligament and the lateral epiphysial vessels are shown. Figure 9—Slice 4. The lateral epiphysial vessels are seen coming from the right in the upper and posterior quadrant of the section. Figure 10—Slice 6. In the upper part of the section on the right the entrance of the lateral epiphysial vessels is shown.

border of the sections: on the right in the right femur (Figs. 10 and 11) and on the left in the left femur (Figs. 14 and 15). The entrance site was found in sections of the lateral part of the femoral head.

The vessels in the femoral neck were constantly situated at its periphery, arising from the cortical shell of the neck and forming a rich network inside the periphery of the femoral neck (Figs. 16 and 20). Anastomoses were less dense and less constant as the centre of the neck was approached (Figs. 17 to 20).

Effect of the position of the nail or screw—The liability to vascular damage by the nail or screw was studied in each of the three positions.

The varus position—A nail or screw inserted in this position seems at first sight likely to cut off the lateral epiphysial vessels, but in these experiments this was never observed. This was because in the varus position the nail tended to be placed in the anterior part of the femoral head—a region not occupied by the lateral epiphysial vessels (Figs. 21 to 23).

The valgus position—This position (Fig. 24) was found to be the one in which interference

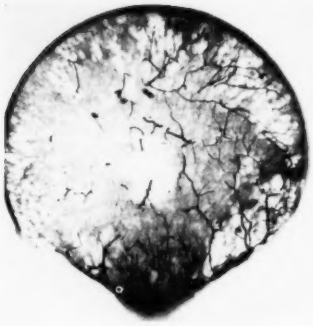


FIG. 12

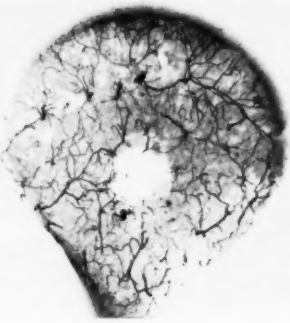


FIG. 13

FIG. 14

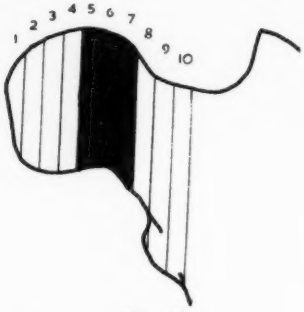


FIG. 15

FIGS. 12 TO 15

Sections through the middle and lateral zones of a left femoral head after injection (slices 5, 6 and 7 as shown in Figure 15). The track of a screw inserted in the " neutral zone " is clearly seen in each section. Figures 12 and 13—Slices 5 and 6. The lateral epiphysial vessels are shown coming from the left in the upper half of the sections. Figure 14—Slice 7. In the left upper zone of the section the entrance of the lateral epiphysial vessels into the head is shown.

FIG. 16

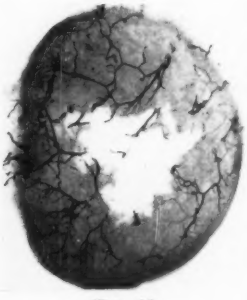


FIG. 18

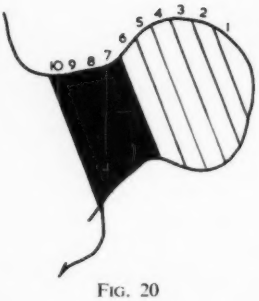


FIG. 20

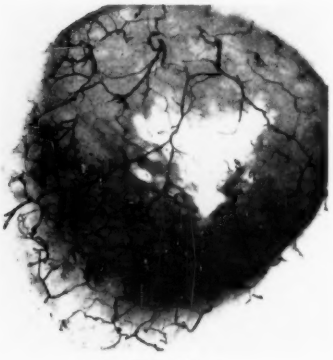


FIG. 17

FIGS. 16 TO 20

Sections through a right femoral neck after injection (slices 7 to 10 as shown in Figure 20). The track of the nail is shown in all four sections. Figure 16—Slice 7. Showing the peripheral vascular network of the neck. Figures 17 to 19—Some of the anastomotic branches crossing the neck have been cut by the nail.

FIG. 19

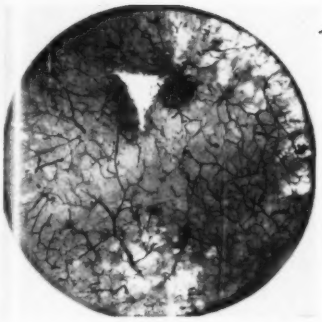


FIG. 21

FIG. 22

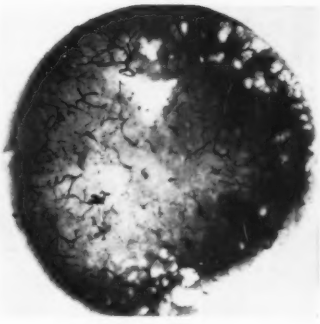


FIG. 23

Sections through the lateral zone of a right femoral head after injection (slices 4 and 5 as shown in Figure 22). A nail has been inserted in the varus position; its track is clearly visible. Figure 21—Slice 4. The nail has passed in front of the lateral epiphysial vessels. Figure 23—Slice 5. There is even more clearance between the nail and the vessels, which are now more peripherally placed.

FIG. 24

Antero-posterior (*left*) and lateral (*right*) radiographs of left femoral head and neck after injection and the insertion of a nail in the valgus position. The tip of the nail is in relation to the lateral epiphysial vessels.

FIG. 25

FIG. 26

Section from specimen shown in Figure 24 (slice 5 as shown in Figure 25). Some of the branches of the lateral epiphysial vessels have been broken by the nail.

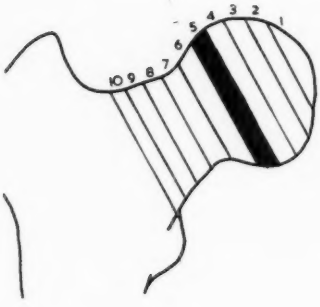


FIG. 27

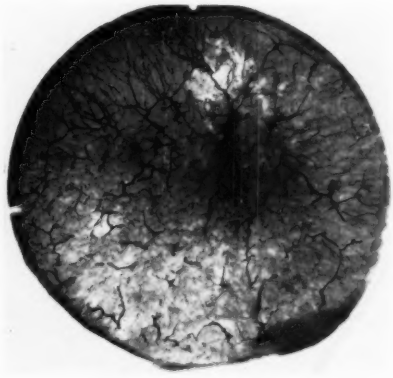


FIG. 28

Section from the right femoral head (slice 5 as shown in Figure 27) of the same individual. The intact lateral epiphysial vessels are well shown, and the extent of the damage caused by the nail can readily be assessed.

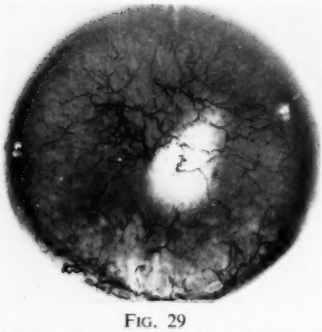


FIG. 29

FIG. 30

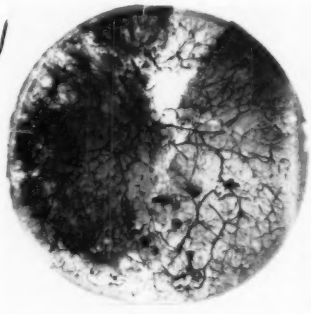


FIG. 31

Sections through the middle and lateral zones of two left femoral heads after injection (slices 3 to 6 in Figure 30 replaced in correct relationship). Figure 29—A screw has been inserted. It has largely avoided damaging the vessels (compare Figure 31). Figure 31—A nail has been inserted. It has damaged several vessels (compare Figure 29).

with the lateral epiphysial vessels was most likely (Figs. 25 to 28). It was the only one in which interference with some of their branches was observed, although in this position too, if the nail were placed anteriorly in the femoral head, the vessels could not be reached (Fig. 2). *The neutral position*—A nail or screw placed in this position could not reach the lateral epiphysial vessels (Figs. 12 to 15), although in some specimens a few anastomoses in the neck were seen to be cut. However, the severing of these anastomoses, which are not constant in any case, seems of secondary importance because of the richness of the peripheral vascular network in the femoral neck.

DISCUSSION

Although the possibility that the nail or screw used in the internal fixation of fractures of the femoral neck may interfere with the blood supply is rather slight, an ideal position for the fixing agent does exist. The possibility of damaging the vessels can be entirely avoided by placing the nail in the zone of the femoral head and neck away from the main vessels responsible for their blood supply. The less favourable zones—the upper and posterior quadrant of the head and the periphery of the neck—should be avoided.

The nearer the nail is placed to the central zone of the femoral head and neck the less will it interfere with the capital blood vessels. This does not apply to the medial third of the head where the vessels coming from the round ligament anastomose with the lateral epiphysial vessels. In this area the vascular anastomoses are so numerous that it is unlikely that the nail however placed could seriously interfere with the blood supply of the femoral head.

No great difference in damage potential between screws and nails was found, although it seems that the screw may be less harmful to the vessels than the three-flanged nail because of its spiral shape and narrower diameter (Figs. 29 to 31).

SUMMARY AND CONCLUSIONS

1. Sixteen injected specimens of human femoral heads and necks, in which a nail or screw had been inserted, were examined.

2. The possibility exists that the fixing agent may interfere with the blood supply of the femoral head. The likelihood of this occurrence is not great.

3. The position of the fixing agent in which vascular damage is least likely is the central area or "neutral zone" of the femoral neck and head.

I wish to express my gratitude to Professor J. Trueta for his constant help and encouragement throughout this work. I am also grateful to Miss M. Litchfield and Mr D. W. Charles for their technical contribution.

REFERENCES

CEDERMARK, J. (1936): Om caputnekroser vid collumfrakturer i spikade fall. *Nordisk Medicinsk Tidskrift*, **11**, 1,044.

HARRISON, M. H. M., SCHAJOWICZ, F., and TRUETA, J. (1953): Osteoarthritis of the Hip: a Study of the Nature and Evolution of the Disease. *Journal of Bone and Joint Surgery*, **35–B**, 598.

HULTH, A. (1956): Intraosseous Venographies of Medial Fractures of the Femoral Neck. *Acta Chirurgica Scandinavica*, Supplementum 214.

JUDET, J., JUDET, R., LAGRANGE, J., and DUNOYER, J. (1955): A Study of the Arterial Vascularization of the Femoral Neck in the Adult. *Journal of Bone and Joint Surgery*, **37–A**, 663.

TRUETA, J., and HARRISON, M. H. M. (1953): The Normal Vascular Anatomy of the Femoral Head in Adult Man. *Journal of Bone and Joint Surgery*, **35–B**, 442.

WATSON-JONES, Sir R. (1955): *Fractures and Joint Injuries*. Fourth edition, Vol. 2, p. 684. Edinburgh and London: E. & S. Livingstone Ltd.

AVASCULAR NECROSIS OF THE FEMORAL HEAD

An Anatomical Study*

T. J. CLAFFEY, SYDNEY, AUSTRALIA

From the Nuffield Orthopaedic Centre, Oxford

Since the recent publication of studies of the vascular pattern of the proximal femoral epiphysis in man by Tucker (1949), Trueta and Harrison (1953), Judet, Judet, Lagrange and Dunoyer (1955), and Trueta (1957) there has been a renewed interest in the mechanism of capital necrosis after fractures of the femoral neck. The literature on the subject is so extensive that no effort will be made here to quote even the most outstanding work, for the aim of this paper is to report the results of an investigation into the vascular damage caused by fractures of the femoral neck in the adult.

ANATOMY

A preliminary study by vascular injection for control material, after the method described by Trueta, Barclay, Franklin, Daniel and Prichard (1947) has provided further information on the findings of Trueta and Harrison (1953). A summary of this work must be made here because we shall repeatedly refer to the findings.

FIG. 1

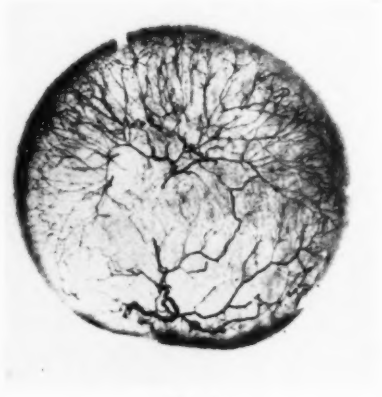


FIG. 2

Figure 1—Sagittal section of an injected femoral head, showing the lateral epiphysial vessels entering above and laterally and passing across the head in a chain to join the ligamentum teres group. Below and medially the inferior metaphysial vessels can be seen entering the bone in several different places. Figure 2—Coronal section of an injected femoral head. Above is part of the vascular chain from the lateral epiphysial vessels to the ligamentum teres. The periphery of the head is supplied in radiating fashion from this chain. Below are the inferior metaphysial vessels. (Reproduced from Trueta and Harrison, by kind permission of the *Journal of Bone and Joint Surgery*.)

Figure 1 shows that in the adult—even, as in this case, shortly after fusion of the growth plate—there is a rich chain of anastomoses between the lateral epiphysial vessels, the ligamentum teres group and the inferior metaphysial vessels. After epiphysial fusion free anastomosis progressively occurs between these vessels and those coming up from the neck and crossing the epiphysial scar. Figure 2 shows a transverse section of the femoral head

* This work was carried out at Oxford while the author was the holder of a Fellowship in Orthopaedic Surgery from the Post-graduate Committee in Medicine of the University of Sydney.

FIG. 3
The femoral neck has been divided without damage to the lateral epiphysial vessels.

FIG. 4
Substantial lateral rotation of the distal fragment tends to relax the tension on the lateral epiphysial vessels instead of rupturing them.

similarly injected. Two main groups of vessels are seen, the lateral epiphysial above and ne inferior metaphysial below. The whole of the periphery of the head is supplied by arbori ng branches from these main vessels.

The relative importance of each group of vessels in supplying the femoral head has b en studied and the part played by the fracture and by the Smith-Petersen nail in causing ischae ia of the head has been investigated.

The vessels of the ligamentum teres—It was found by Trueta and Harrison (1953) that ne vessels of the ligamentum teres remained patent in the aged. This was confirmed in all ne injected specimens prepared for this work. However, the fact that the vessel is patent d es not prove that it is capable of keeping the head alive when all other sources of blood sup ly are interrupted. It was impossible, clinically, to detect whether the ligamentum teres as intact or not; consequently, attention was concentrated on the other two sources of blo d, the inferior metaphysial and the lateral epiphysial vessels.

FIG. 5

FIG. 6

Figure 5—Lateral view showing the lateral epiphysial vessels before displacement. Figure 6—After the distal fragment has been considerably displaced backwards.

Inferior metaphysial vessels—These vessels lying on the antero-inferior aspect of the femoral neck are certainly torn in all displaced fractures of the femoral neck by the lateral rotation of the distal fragment; in fractures produced experimentally this was always so. The extensive anastomosis of the inferior metaphysial vessels with the surrounding vascular groups may explain why their damage is not important.

No constant relationship between the integrity of this group of vessels and subsequent avascular necrosis could be found from radiological evidence.

The lateral epiphysial vessels—In the vascular studies on children by Trueta (1957) it was found that the lateral epiphysial vessels were responsible for the blood flow of almost the whole of the femoral capital epiphysis until the age of six to eight years and that from that age onwards the vessels from the round ligament supplied a variable segment of the medial part of the head. Trueta and Harrison (1953) found that a large anastomosis between the lateral epiphysial vessels and those of the round ligament persisted indefinitely and that the most conservative estimate made the lateral epiphysial vessels responsible for the nourishment of at least the outer two thirds of the femoral head.

In the present work we investigated afresh the outer course of the lateral epiphysial vessels before cutting the bone.

Two important findings were made in necropsy specimens in which we caused experimental fractures after intravascular perfusion. The first observation was that a fracture distal to the point of entry of these vessels into the bone caused, as in accidental fractures in the living subject, upward displacement and lateral rotation of the distal fragment. When this happened the superior retinaculum containing the lateral epiphysial vessels stripped off the bone and a considerable displacement was possible without damage to, or tension of, the vessels (Figs. 3 to 7). Indeed, with this usual displacement the lateral epiphysial vessels were relaxed rather than stretched. It was found that only when the distal fragment was displaced proximally by a distance equal to half the diameter of the head did any tension begin to be exerted on the vessels.

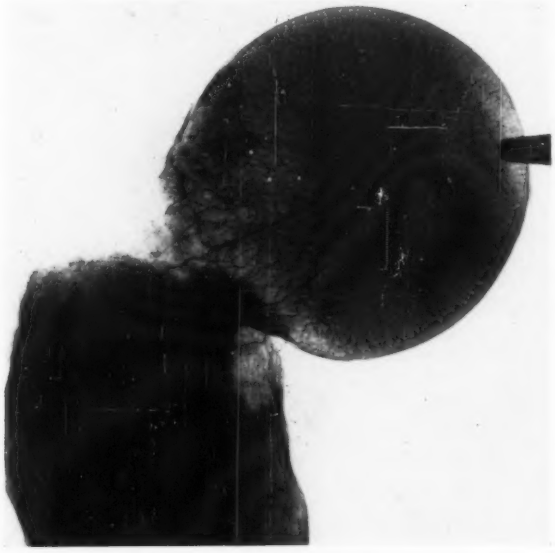


FIG. 7
Extreme rotation of the head backwards fails to damage the lateral epiphysial vessels.

The other interesting observation refers to the course of the lateral epiphysial vessels within the bone. These vessels enter the femoral head at the point where the neck turns abruptly upwards, about half a centimetre below the margin of the articular cartilage. Occasionally they enter distal to this point but seldom proximal to it. We shall refer later to the importance of these vessels in keeping the femoral head alive.

In all our specimens, when the line of fracture reached the point where the neck turned upwards, the lateral epiphysial vessels were found completely severed. This was to be expected from the work of Trueta and Harrison; it makes the determination of the line of fracture a necessity before prognosis is made.

RADIOGRAPHIC STUDY OF THE FRACTURE LINE

The pre-reduction radiographs of fractures gave no reliable information as to whether the lateral epiphysial vessels had been damaged or not, for the exact site of the fracture could not be determined until radiographs were obtained after reduction, and even then the line of fracture was often doubtful.

For this study fifty unselected radiographs after reduction of fractures of the femoral neck were examined. It was found that, of these fifty cases, in only twelve was the fracture line

clearly seen to cross the interosseous course of the lateral epiphysial vessels. Figures and 9 are two examples of this group. In none of the other cases could enough informatic be gathered from the radiographs about the exact site of the fracture. It was thus doubtf whether, with the present radiographic technique, the integrity or otherwise of the later epiphysial vessels could be determined.

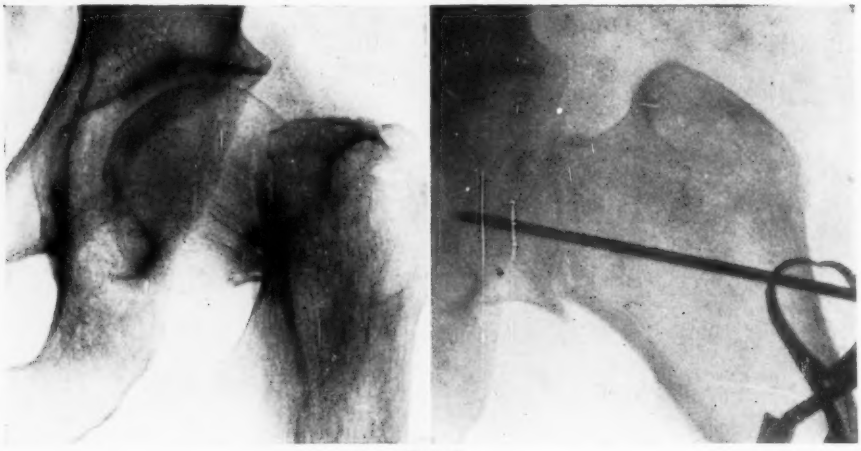


FIG. 8
Example of group 1 fracture before (*left*) and after (*right*) reduction.

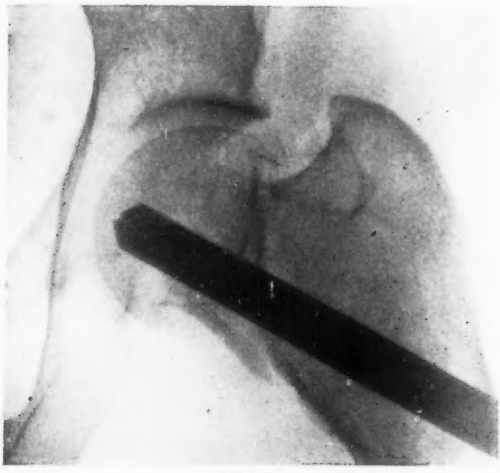


FIG. 9
Another example of group 1 fracture. In this case a loose fragment is present at the critical point. This is always a dangerous state of affairs.

THE SMITH-PETERSEN NAIL

In order to investigate the relationship between the Smith-Petersen nail and the vessels of the femoral head twelve necropsy specimens from subjects over the age of sixty-five were injected with Micropaque by the technique of Trueta and Harrison (1953). A Smith-Petersen nail was then inserted, and the specimen was radiographed, decalcified and studied in

Spalteholz preparations. Routine radiographs gave the impression that a nail placed in the supero-lateral aspect of the head might sever the anastomosis between the lateral epiphysial vessels and those of the ligamentum teres.

Closer three-dimensional study of the Spalteholz preparations reveals an important point (Figs. 10 and 11). The nail is seen to sever one branch of the main lateral-medial anastomosis but not both. This was found in all specimens. No matter where the nail was placed it was impossible to sever completely these vascular connections. In lateral views it was seen that the width of the nail was about the same as the width of the vascular leash forming the anastomosis; so it was virtually impossible to place the nail in such a position that it could completely sever this main vascular pathway.

A nail driven through the region of the attachment of the ligamentum teres might be expected to interrupt the proximal part of the great transverse vascular anastomosis. To investigate this possibility a further six uninjected specimens were studied. A Smith-Petersen nail was placed in each case, passing deep into this part of the femoral head. The circumflex vessels on the back of the neck were then injected with a mixture of 3 per cent Micropaque and 2 per cent Berlin Blue. It was expected that the flow through the ligamentum teres group would be stopped if the nail had severed the anastomotic chain. The result was the same in all instances. The dye passed along the anastomosis and emerged from the cut end of the ligamentum teres.

These findings suggest that it is almost impossible to interrupt seriously the anastomosis between the vessels of the lateral epiphysial group and those of the ligamentum teres with a Smith-Petersen nail. It seems most unlikely therefore that the nail contributes greatly to avascular necrosis of the femoral head.

THE ROLE OF THE LATERAL EPIPHYSIAL VESSELS IN AVASCULAR NECROSIS

Clinical evidence—Consideration of these observations suggested that the most likely cause of avascular necrosis of the femoral head was damage to the lateral epiphysial vessels. To collect further information on this point records of the femoral neck fractures seen in 1950–55 in the Accident Service of the Nuffield Orthopaedic Centre at the Radcliffe Infirmary were studied. These were classified by their radiographic appearance into two groups: 1) those in which the fracture line undoubtedly reached the point of entry of the lateral epiphysial vessels; and 2) all the other cases.

Of a total of 197 patients 178 could be traced. The radiographs of twenty-four of these clearly belonged to group 1. *In all of them avascular necrosis of the femoral head occurred.* Of a further group of twenty cases in which the radiographs were not clear enough to disclose the integrity or otherwise of the area of the lateral epiphysial vessels, ten suffered from necrosis of the head.

DISCUSSION

It was impossible from this investigation to ascertain whether damage to the vessels of the round ligament played any part in the causation of capital necrosis. The fact that necrosis occurred in all the cases in which the fracture line crossed the area of penetration of the lateral epiphysial vessels suggests that the damage to these vessels is the most important single factor responsible for necrosis of the femoral head.

The finding of the part played by these vessels in the prognosis of femoral neck fractures may suggest a new interpretation to the observation by Pauwels (1935) that the more vertical was a fracture of the femoral neck the less likely was it to end successfully. That the vertical fractures are more unstable and more likely to slip seems obvious. However, this can bear only on the rate and quality of consolidation but not on the vascular integrity of the femoral head. Figure 12 shows schematically that an oblique or transverse fracture can interfere with

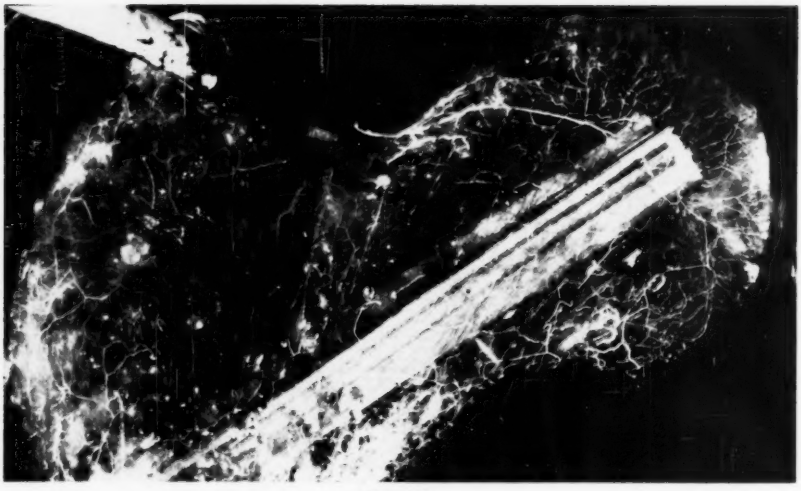


FIG. 10

Spalteholz preparation of an injected femoral head and neck viewed from in front. A nail has been inserted, and one fin can be seen cutting through the vascular chain.

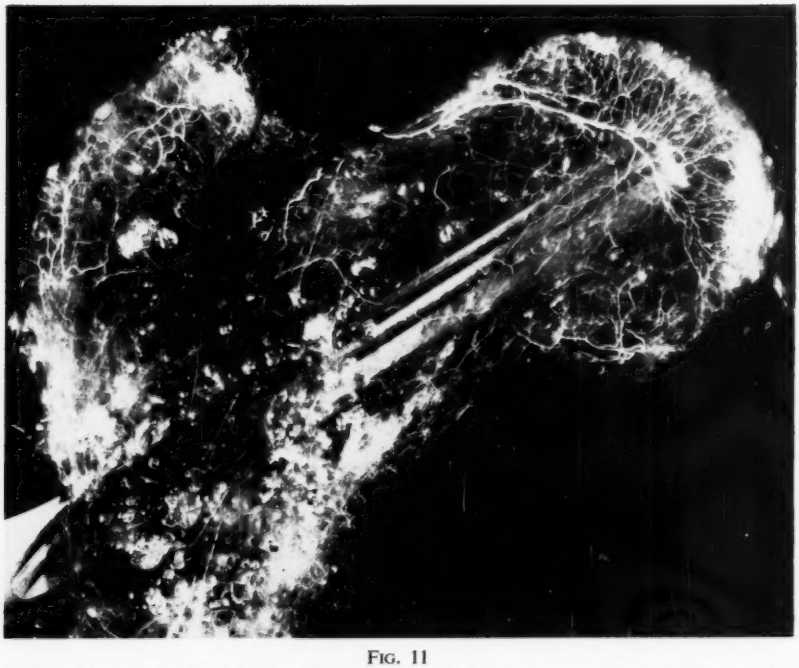


FIG. 11

Same specimen as in Figure 10 viewed from behind. Note that the posterior part of the vascular chain is intact.

the lateral epiphysial vessels as much as a vertical fracture if it is placed at the proximal end of the neck.

Two other observations made are worth emphasising. One, which we found surprising, was that displacement of the distal fragment hardly damaged the lateral epiphysial vessels. The other observation was the limited part that the nail plays in causing vascular interference sufficient to endanger the vitality of the femoral head.

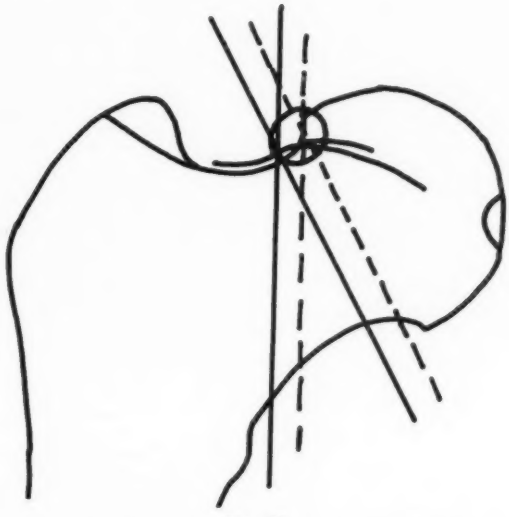


FIG. 12
The interrupted lines represent group 1 fractures of varying obliquity.

It may be concluded from this work that determination of the line of fracture gives the best indication of the fate of the femoral head.

I am indebted to Professor J. Trueta for his inspiration and guidance in this work. The clinical material from the Accident Service of the Radcliffe Infirmary was made available by the kind permission and cooperation of Mr J. C. Scott. To him and his colleagues, Mr R. G. Taylor and Mr J. D. Morgan, I express my gratitude. I also thank Mr A. Mann and Mr D. W. Charles of the technical staff of the Nuffield Department of Orthopaedic Surgery for their help.

REFERENCES

JUDET, J., JUDET, R., LAGRANGE, J., and DUNOYER, J. (1955): A Study of the Arterial Vascularization of the Femoral Neck in the Adult. *Journal of Bone and Joint Surgery*, **37–A**, 663.

PAUWELS, F. (1935): *Der Schenkelhalsbruch, ein mechanisches Problem.* Stuttgart: Enke.

TRUETA, J. (1957): The Normal Vascular Anatomy of the Human Femoral Head During Growth. *Journal of Bone and Joint Surgery*, **39–B**, 358.

TRUETA, J., BARCLAY, A. E., FRANKLIN, K. J., DANIEL, P. M., and PRICHARD, M. M. L. (1947): *Studies of the Renal Circulation.* Oxford: Blackwell Scientific Publications.

TRUETA, J., and HARRISON, M. H. M. (1953): The Normal Vascular Anatomy of the Femoral Head in Adult Man. *Journal of Bone and Joint Surgery*, **35–B**, 442.

TUCKER, F. R. (1949): Arterial Supply to the Femoral Head and its Clinical Importance. *Journal of Bone and Joint Surgery*, **31–B**, 82.

A STUDY OF THE MECHANICS OF SPINAL INJURIES

ROBERT ROAF, OSWESTRY and LIVERPOOL, ENGLAND

The basic spinal unit consists of two intact vertebrae joined by an intervertebral disc, two posterior articulations and a number of ligaments (Fig. 1). There have been a number of studies of isolated intervertebral discs (Virgin 1951; Brown, Hansen and Yorra 1957) but there are few published accounts of the reactions of a complete spinal unit when subjected to a variety of forces. This paper describes the results of studies of such spinal units when subjected to forces of different magnitude and direction—compression, flexion, extension, lateral flexion, rotation and horizontal shear.

In particular I have been interested in the question of the discrepancy between the magnitude of the force and the resulting lesion. In other words, why, on one occasion, does a relatively small force produce a serious dislocation or major disc prolapse, whereas, on another occasion, a much larger force produces only a relatively minor vertebral crush? I believe that it is now possible to understand the underlying reasons for these differences and to reproduce all varieties of spinal injury experimentally by accurately controlled forces.

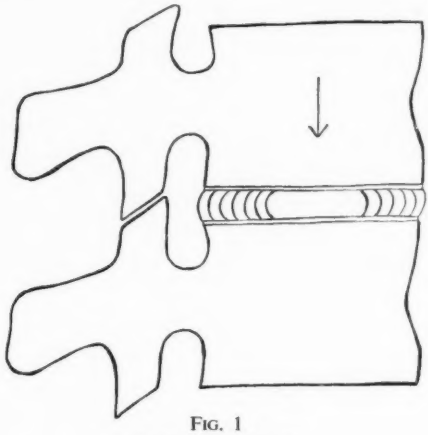


FIG. 1
A single spinal unit.

Definitions—A force has direction and magnitude. A given tissue—bone or ligament—breaks when sufficient force is applied in the right direction to a localised area for a given time. The meaning of compression force is obvious; distraction is nearly as clear, though it is a pity that the more exact term extension has been wrongly applied to backward flexion. By flexion and extension I mean rotation in the sagittal plane about the normal axis of rotation; thus, in flexion there is relaxation of the anterior ligaments and tightening of the posterior ligaments. Hyperflexion means disruption of the posterior elements by a distracting force in the sagittal plane. Extension and hyperextension are the opposite. Rotation force is a couple applied in the horizontal plane; in practice rotation and horizontal shear forces are usually combined.

MATERIAL AND METHODS

Portions of healthy cervical, thoracic and lumbar spines were obtained, mostly from children and young adults, as soon after death as possible. The cause of death was either injury or acute non-spinal infection. In a few cases specimens from older patients were used,

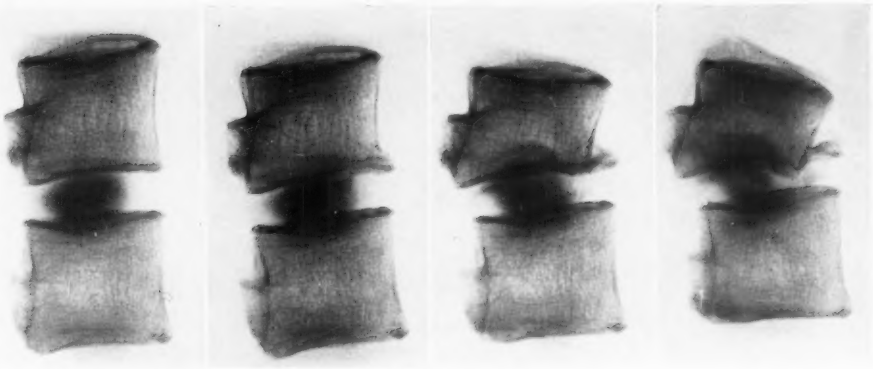


FIG. 2
Normal discographs under increasing pressure.

either to investigate the effect of compression in spinal units in which the nucleus pulposus had become fibrotic, or to investigate the effect of rotation forces upon an arthritic cervical spine. The specimens were at once placed in a " deep-freeze." Before use they were immersed in warm water for twelve to twenty-four hours. For certain experiments the nucleus pulposus was injected with a 20 per cent solution of potassium iodide made to suitable viscosity with glycerine and sufficient methylene blue to produce the colour, thus enabling the nucleus to be studied both radiographically (Fig. 2) and visually. This injection did not appear to alter the mechanical properties of the disc; in other words there was no observed difference between injected and uninjected specimens.

For ease of analysis the forces were usually applied slowly; for example, compression forces were applied with a standard Denniston testing machine. It is realised that most human injuries are the consequence of rapidly acting forces, but on the few occasions when a rapidly acting force was used the general pattern of response was similar to the pattern when slowly acting forces were used.

RESULTS

COMPRESSION FORCES

Intact disc and vertebrae—Provided the nucleus pulposus retains its normal turgor (Petter 1933), there is a uniform pattern of response seen most characteristically in the lumbar region where the nuclei pulposi are relatively larger and their reactions can be studied more easily.

As vertical pressure increases there is a very slight bulge of the annulus but no alteration in the shape of the nucleus pulposus, and the major distortion is a bulge of the vertebral end-plate. Indeed it is only as this happens that the two vertebrae move closer to each other (diminution of disc space); the annular bulge is secondary to this and is not due to pressure from the nucleus pulposus. (The annular bulge is more marked when there is no nucleus pulposus, just as an under-inflated pneumatic tyre bulges more than a well-inflated one.)

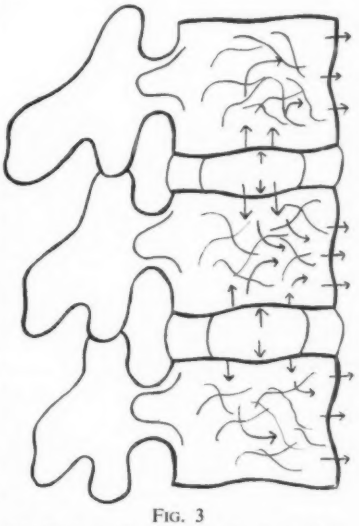


FIG. 3
Mechanism of spinal shock absorption. Arrows show direction of pressure gradients and fluid movements.

Bulging of the end-plate causes blood to be squeezed out of the cancellous bone of the vertebral body. This is the first sign of increasing compression and is accompanied by a fall in pressure (Fig. 3). It is considered that this is an important shock-absorbing mechanism in the spine. As pressure increases the end-plate bulges more and finally cracks (Figs. 4 to).

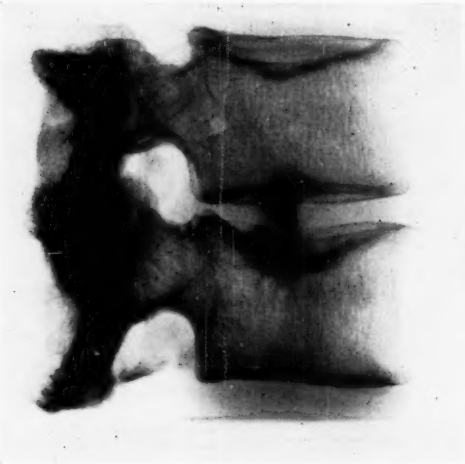


FIG. 4

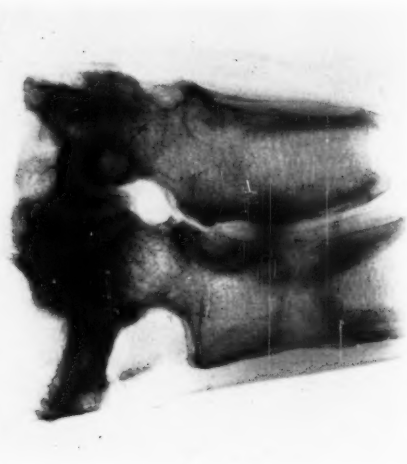


FIG. 5

Figure 4—Bulging of end-plate. Figure 5—Vertical split in body. Nucleus pulposus undeformed.

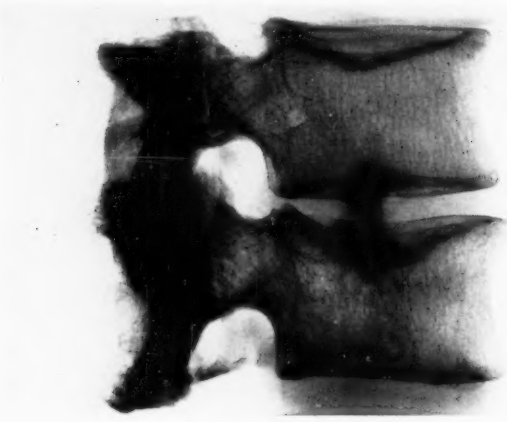


FIG. 6

End-plate fracture. Nucleus undeformed.

Up to this point there has been relatively little bulging of the annulus. Once the end-plate has been broken, nuclear material is displaced into the vertebral body (Figs. 7 and 8). The disc has now lost its turgor and the annulus begins to bulge like the walls of a flat tyre. Continued pressure leads to disintegration of the vertebral body. If the line of force is oblique the line of fracture is oblique (Fig. 9).

Figure 10 shows typical distance-pressure graphs for complete spinal units and for isolated wet and dry vertebrae. The results have been uniform in eighteen experiments: 1) the

intact disc is more resistant to vertical compression than the vertebral body; 2) on compression of an intact spinal unit the vertebral end-plate bulges; 3) this causes oozing of blood from the vertebral body, which appears to be a major shock-absorbing mechanism (Fig. 3); 4) there is little, if any, movement or distortion of the normal nucleus pulposus.

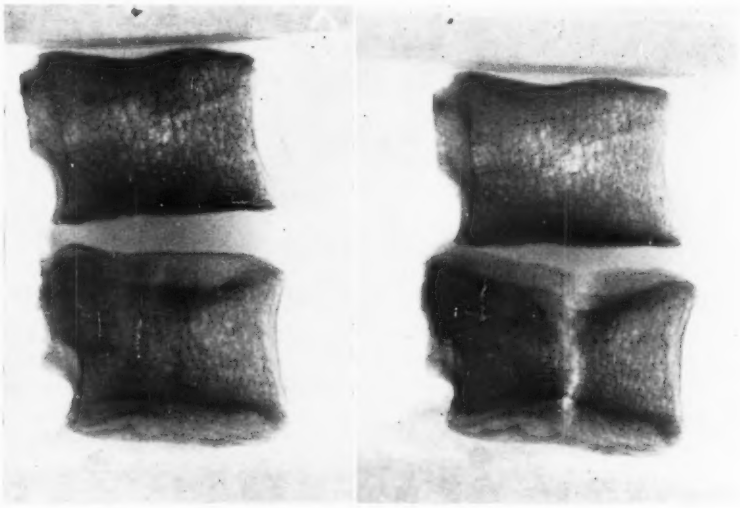


FIG. 7
End-plate fracture leading to vertical fracture of body.

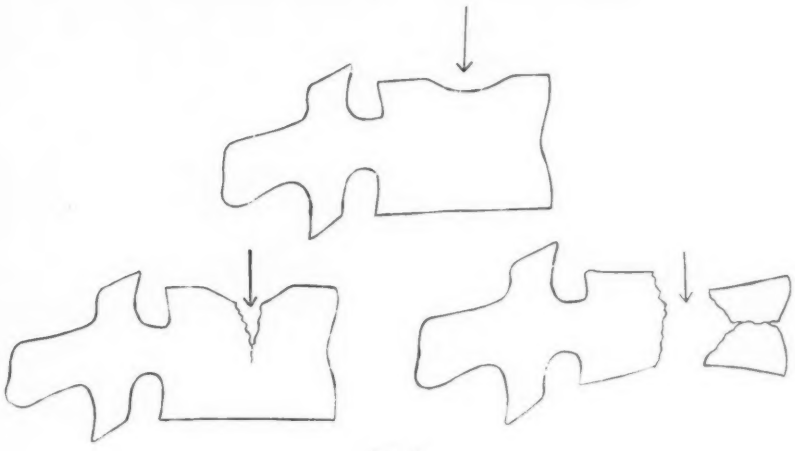


FIG. 8
Diagrams of sequence of events.

There is no significant bulging of the annulus until the nucleus has lost its turgor in consequence of nuclear displacement into the vertebral body from collapse of the vertebral end-plate.

There was never any evidence of nuclear material pressing upon the annulus and causing a localised protrusion, as has been often suggested (Armstrong 1952). Indeed, if the annulus

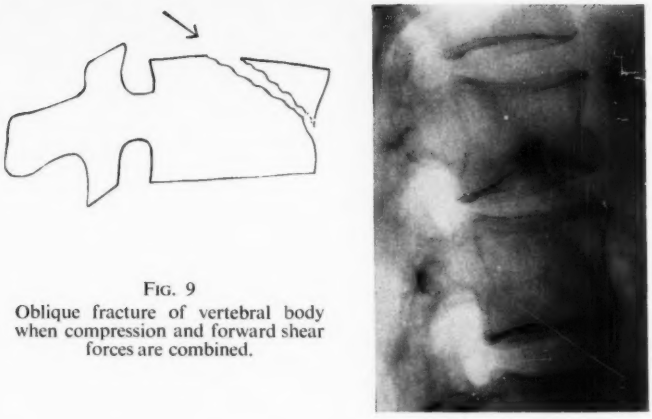


FIG. 9
Oblique fracture of vertebral body when compression and forward shear forces are combined.

is artificially weakened at a given point, nuclear fluid exudes and, being fluid, is dispersed evenly over the surface. There is no annular bulge until the whole disc disintegrates, and the site of the bulge is not determined by the site of annular weakness and nuclear seepage. Disc protrusions, as observed clinically, consist of annular material. Indeed how could freely flowing fluid cause a localised nerve root compression?

Compression of specimens from older subjects in which the nucleus was no longer fluid, and asymmetrical compression, produced a different form of fracture. Pressure under these conditions is mainly transmitted through the annulus and the result is either tearing of the annulus, general collapse of the vertebrae due to buckling of the sides (Fig. 11) or a marginal plateau fracture (Figs. 12 and 13).

Compression of denucleated spinal units—The healthy nucleus pulposus of a child or young adult in life is a viscous liquid very similar to the contents of a ganglion. Its shape is maintained by the surrounding walls of the cavity in which it lies—vertebral end-plate and annulus fibrosus. The mechanical properties of the intervertebral disc depend on a normal nuclear

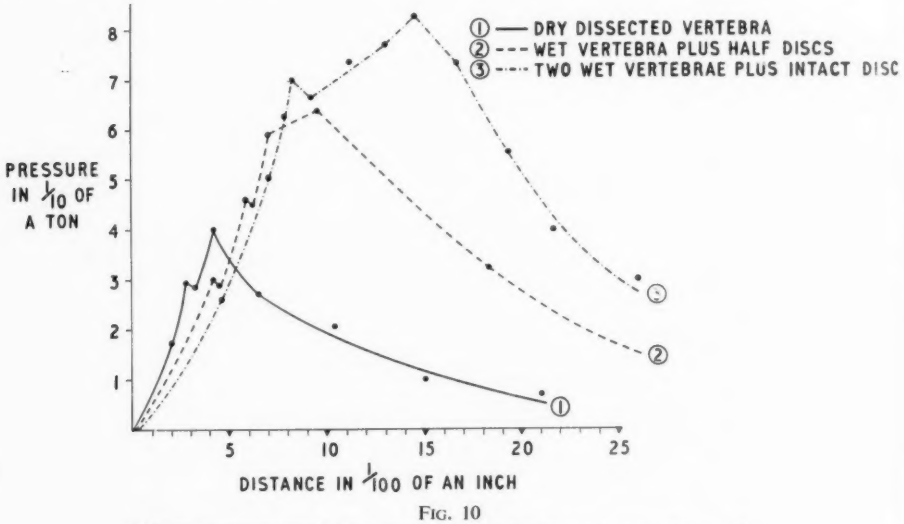


FIG. 10
Distance-pressure graphs for a complete spinal unit, a wet and a dry vertebra.

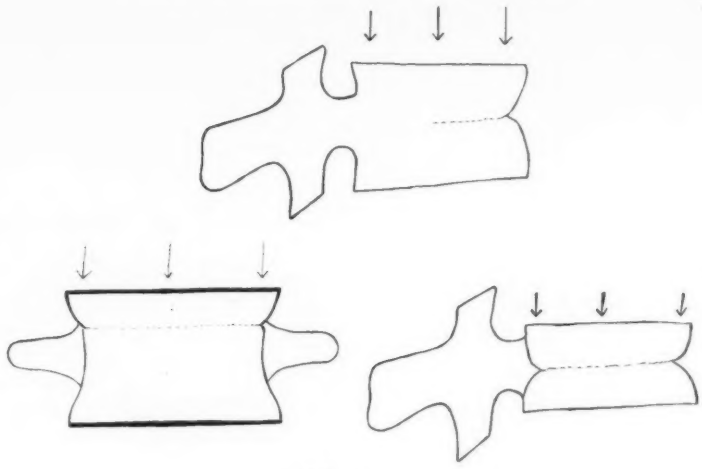


FIG. 11
General collapse of the vertebral body.

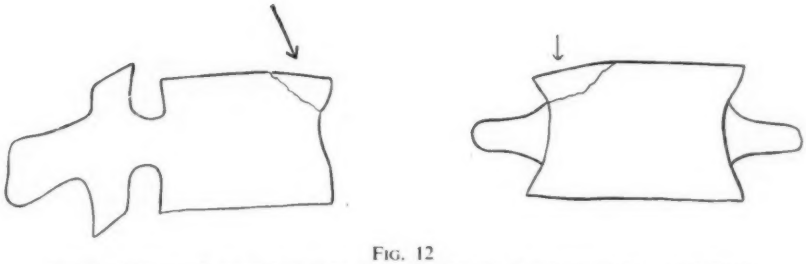


FIG. 12
Marginal plateau fracture (not to be confused with the slice fracture due to rotation).

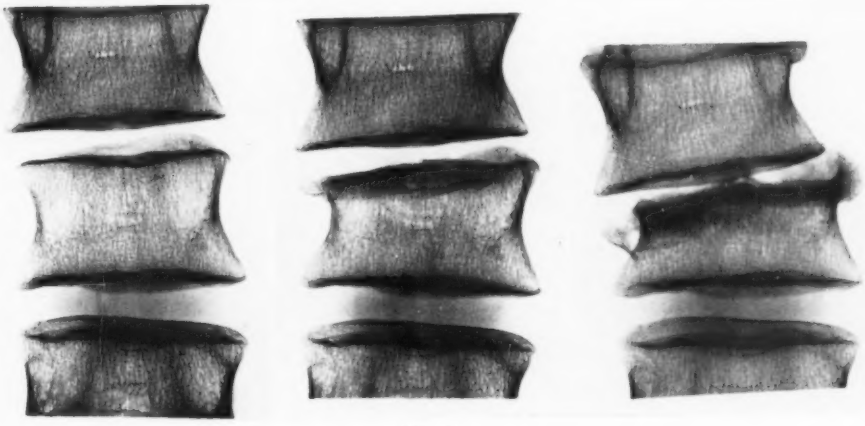


FIG. 13
Marginal plateau fracture.

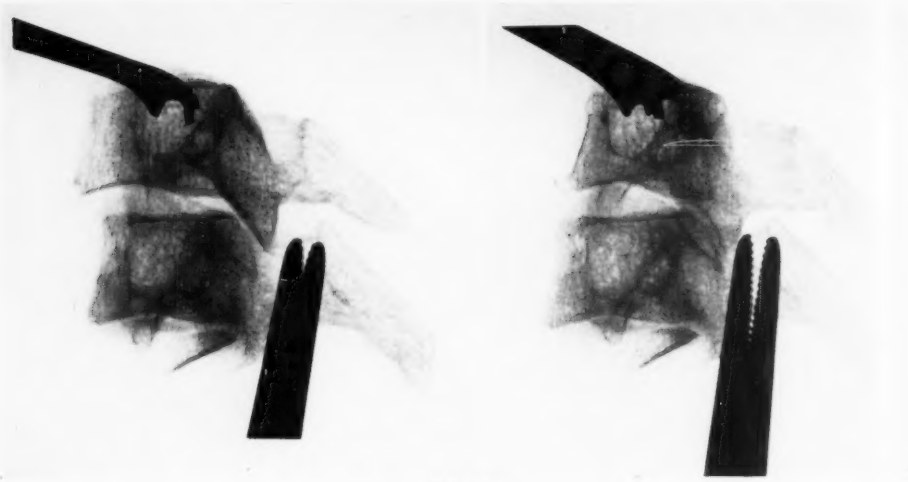


FIG. 14
Abnormal mobility after enucleation.

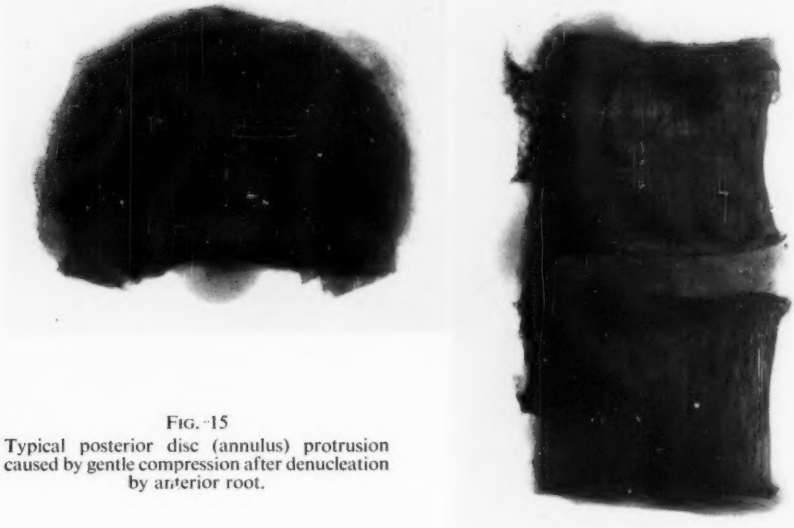


FIG. 15
Typical posterior disc (annulus) protrusion caused by gentle compression after denucleation by anterior root.

turgor. The disc can be compared to a plant cell. If the nucleus is normally hydrated it has turgor and retains its shape. If the nucleus is dehydrated (mechanically or otherwise) it collapses and loses its normal mechanical properties. If intranuclear pressure increases the evidence is that the vertebral end-plate bulges: this may be an important cause of back pain.

After mechanical removal of the fluid nucleus pulposus there is abnormal mobility between the vertebral bodies (Roaf 1958) (Fig. 14). If such a " denucleated " unit is compressed the result is quite different. The annulus at once bulges, there is no bulging of the vertebral end-plate and no oozing of blood from the vertebral body. If the compression is continued, typical disc (annular) prolapses, as seen at operation, are produced (Figs. 15 and 16).

The vertebral end-plate fracture has considerable medico-legal importance. A patient may easily sustain a compression injury without a radiographically visible fracture. Nevertheless, he may have sustained a vertebral end-plate fracture with intracorporeal nuclear protrusion and loss of disc turgor. His disc is now vulnerable, and a short time later a relatively minor injury may cause a severe and incapacitating annular prolapse for which the first, not the second, injury was the real cause.

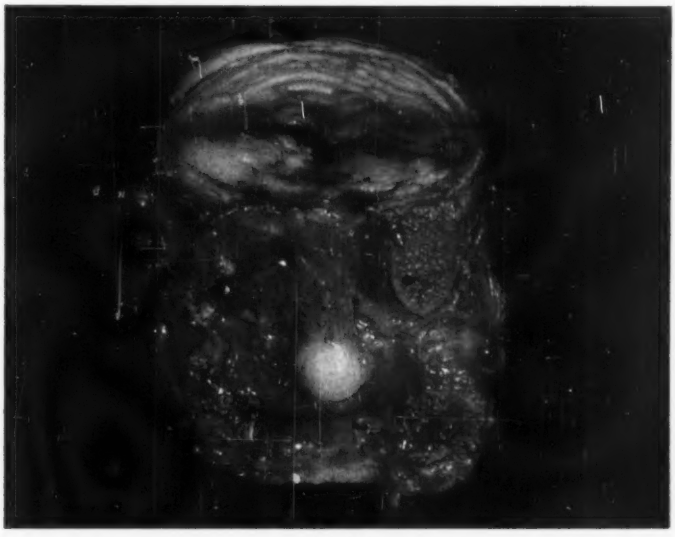


FIG. 16
Photograph of specimen the radiographs of which are shown in Figure 15.

FLEXION FORCES

I have never succeeded in producing pure hyperflexion injuries of a normal intact spinal unit. Before the posterior ligaments rupture, the vertebral body always becomes crushed. This is what one would expect from an elementary calculation of moments. Pressure at the fulcrum must be three to four times as great as tension on the posterior ligaments, whose breaking point is about 400 lb. (Brown *et al.* 1957). Pressure on the nucleus is therefore 1,200–1,600 lb., which exceeds the breaking point of the vertebral end-plate.

There was never any significant alteration in either the position or the shape of the healthy nucleus pulposus (Figs. 17 to 22) or any significant annular bulge. If any bulge occurs it is always posteriorly on extension and anteriorly on flexion. Similarly, after denucleation annular bulging occurs anteriorly on forward flexion, not posteriorly as is often suggested.

EXTENSION AND LATERAL FLEXION

Very similar results were obtained with extension and lateral flexion (Figs. 17 to 22). It was found impossible to rupture the anterior longitudinal ligament of the cervical spine by a pure hyperextension force: crush fracture of the neural arch occurred first. Here again there was no detectable alteration in shape or position of the nucleus pulposus and no significant annular bulge posteriorly on extension. After denucleation there was excessive mobility and an annular bulge posteriorly on extension.

ROTATION

The intervertebral discs, joints and ligaments were found to be very resistant to compression, distraction, flexion and extension, but very vulnerable to rotation and horizontal

shearing forces. In the cervical region particularly, manual rotation will produce tearing f ligaments and dislocation. If the spine is held in slight flexion and a rotation force is appli d the posterior ligaments, joint capsules and posterior longitudinal ligament tear in that ord , and a typical dislocation results (Figs. 23 and 24). This may or may not be associated wit a

FIG. 17

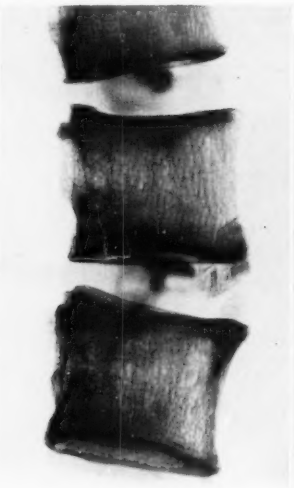


FIG. 18

FIG. 19

FIG. 20

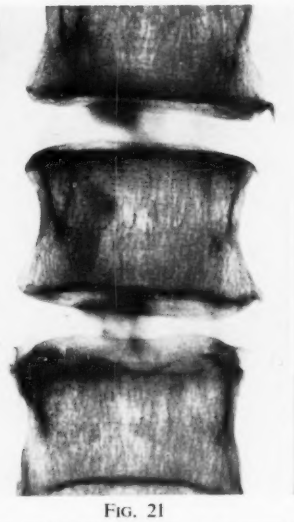


FIG. 21

FIG. 22

Nucleographs of spine in hyperflexion and hyperextension. The neural arches have been removed to increas the range of movement. Even so, there is hardly any change in shape or position of the nucleus pulposus. Figure 17—Flexion. Figure 18—Extension. Figure 19—Hyperflexion. Note avulsion of end-plate. Figure 20— Extension. Figures 21 and 22—Lateral flexion.

fracture of the vertebral body depending on the amount of associated compression forc . It is clear that the common clinical appearances of a cervical dislocation or fracture-dislocatio usually attributed to flexion or hyperflexion are really due to rotation (Figs. 23 and 24). Similarly, it has not been possible to rupture the anterior longitudinal ligament b

FIG. 23

FIG. 24

Figure 23—Dislocation produced by gentle rotation force. Figure 24—The same specimen after reduction.

FIG. 25

FIG. 26

Figure 25—Rupture of anterior longitudinal ligament produced by rotation force. Figure 26—The same specimen after reduction.

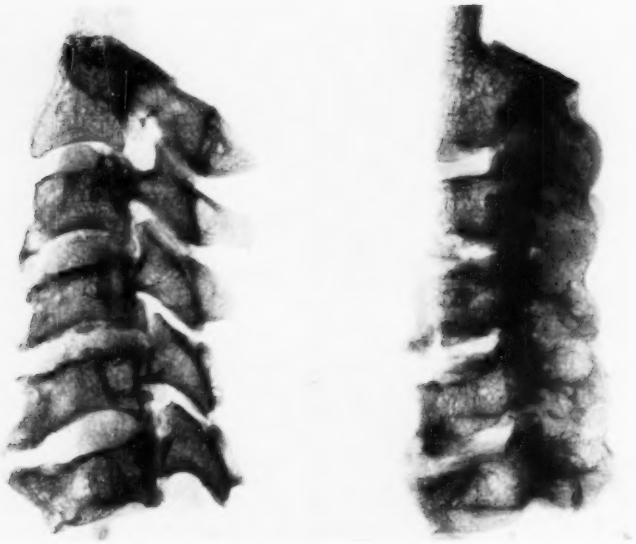


FIG. 27 FIG. 28

Rotation force applied to arthritic spine. Figure 27—Lateral view: no fracture seen. Figure 28—Oblique view, showing marked displacement with vertical fracture of the vertebral body. Anterior longitudinal ligament appears intact

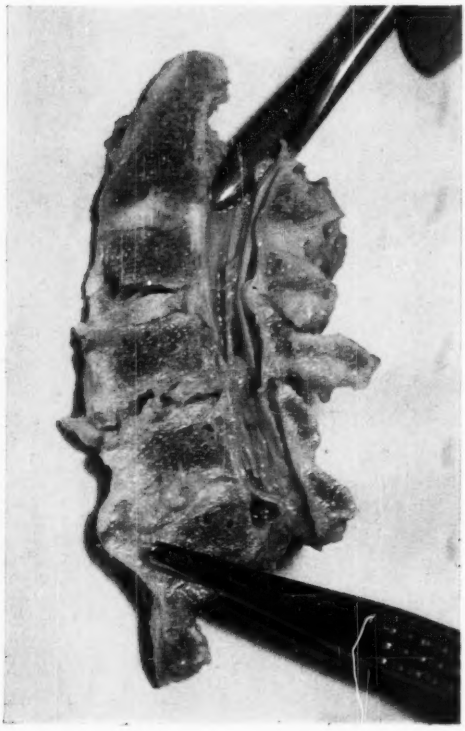


FIG. 29
Photograph of specimen after rotation injury.

FIG. 30

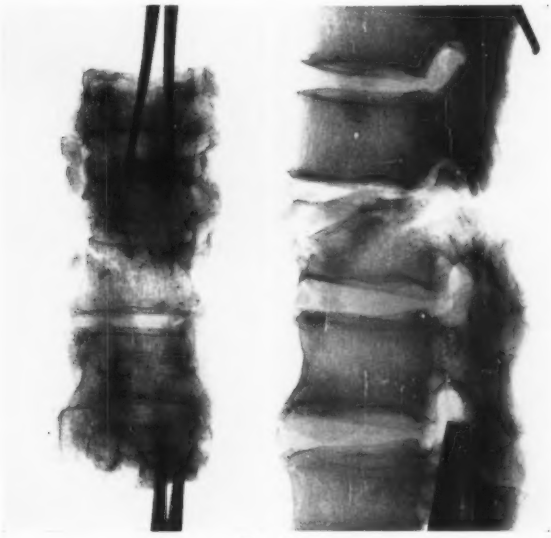


FIG. 31

Figure 30—Rotation injury of thoraco-lumbar region. Figure 31—The same specimen, closed.

FIG. 32

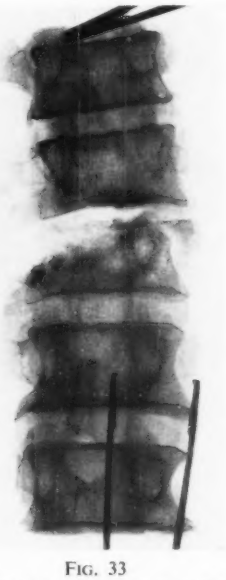


FIG. 33

FIG. 34

Figure 32—Rotation injury, open. Figure 33—Same specimen, closed. Figure 34—Another rotation injury.

hyperextension; the neural arches always fracture first. If the spine is rotated in the extended position the anterior longitudinal ligament easily ruptures (Figs. 25 and 26). The so-called hyperextension injury is really a rotation injury. If the nucleus pulposus has lost its turgor these effects are produced more easily, and rotation and horizontal shear forces will produce considerable intervertebral displacement or subluxation even without rupture of the anterior longitudinal ligament (Figs. 14, 27 to 29). Similarly, in the thoraco-lumbar region a relatively small rotation force will produce a dislocation (Figs. 30 to 36). If the spine is held in flexion

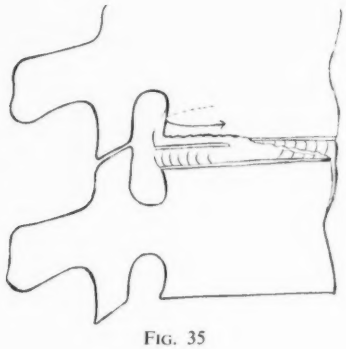


FIG. 35

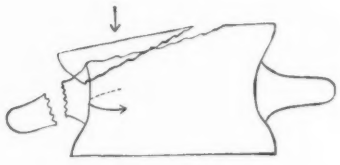


FIG. 36

FIGS. 35 AND 36

Figure 35—Diagram of pure rotation injury. Figure 36—Diagram of rotation combined with slight compression. This injury is unstable and must be differentiated from the stable marginal plateau fracture. (Compare Figure 12.)

this can occur without fracture of the articular processes, although it is more common to have an associated articular process fracture. Holdsworth and Hardy (1953) have pointed out that spontaneous reduction may occur and that radiological examination alone may fail to reveal the extent of the damage and the degree of spinal instability (Figs. 30 to 36). My clinical and experimental findings confirm their views on this point.

SUMMARY

1. Compression forces are mainly absorbed by the vertebral body. The nucleus pulposus, being liquid, is incompressible. The tense annulus bulges very little. On compression the vertebral end-plate bulges and blood is forced out of the cancellous bone of the vertebral body into the perivertebral sinuses. This appears to be the normal energy-dissipating mechanism on compression.

2. The normal disc is very resistant to compression. The nucleus pulposus does not alter in shape or position on compression or flexion. It plays no active part in producing a disc prolapse. On compression the vertebral body always breaks before the normal disc gives way. The vertebral end-plate bulges and then breaks, leading to a vertical fracture. If the nucleus pulposus has lost its turgor there is abnormal mobility between the vertebral bodies. On very gentle compression or flexion movement the annulus protrudes on the concave aspect—not on the convex side as has been supposed.

3. Disc prolapse consists primarily of annulus; it occurs only if the nucleus pulposus has lost its turgor. It then occurs very easily as the annulus now bulges like a flat tyre.

4. I have never succeeded in producing rupture of normal spinal ligaments by hyperextension or hyperflexion. Before rupture occurs the bone sustains a compression fracture. On the other hand horizontal shear, and particularly rotation forces, can easily cause ligamentous rupture and dislocation.

5. A combination of rotation and compression can produce almost every variety of spinal injury. In the cervical region subluxation with spontaneous reduction can be easily produced by rotation. If disc turgor is impaired this may occur with an intact anterior longitudinal

ligament and explains those cases of tetraplegia without radiological changes or a torn anterior longitudinal ligament. The anterior longitudinal ligament can easily be ruptured by a rotation force and in my experience the so-called hyperextension and hyperflexion injuries are really rotation injuries.

6 Hyperflexion of the cervical spine or upper thoracic spine is an anatomical impossibility. In all spinal dislocations a body fracture may or may not occur with the dislocation, depending upon the degree of associated compression. In general, rotation forces produce dislocations, whereas compression forces produce fractures.

I would like to express my thanks to Mr W. G. Davies, Superintendent Radiographer at the Robert Jones and Agnes Hunt Orthopaedic Hospital, for invaluable assistance in these experiments.

REFERENCES

ARMSTRONG, J. R. (1952): *Lumbar Disc Lesions.* Edinburgh and London: E. & S. Livingstone Ltd.

BROWN, T., HANSEN, R. J., and YORRA, A. J. (1957): Some Mechanical Tests on the Lumbosacral Spine with Particular Reference to the Intervertebral Discs. *Journal of Bone and Joint Surgery*, **39-A,** 1,135.

HOLDSWORTH, F. W., and HARDY, A. (1953): Early Treatment of Paraplegia from Fractures of the Thoracolumbar Spine. *Journal of Bone and Joint Surgery*, **35-B,** 540.

PETTER, C. K. (1933): Methods of Measuring the Pressure of the Intervertebral Disc. *Journal of Bone and Joint Surgery*, **15,** 365.

ROAF, R. (1958): The Treatment of Resistant Scoliosis. *Proceedings of the Royal Society of Medicine* (Section of Orthopaedics), **51,** 237.

VIRGIN, W. J. (1951): Experimental Investigations into the Physical Properties of the Intervertebral Disc. *Journal of Bone and Joint Surgery*, **33-B,** 607.

CELL DIVISION IN ENDOCHONDRAL OSSIFICATION

A Study of Cell Proliferation in Rat Bones by the Method of Tritiated Thymidine Autoradiography

N. F. KEMBER, LONDON, ENGLAND

From the Department of Physics, the Institute of Cancer Research, Royal Cancer Hospital, London

The use of tritiated thymidine, associated with high resolution autoradiographic techniques offers many opportunities for studying the proliferation and fate of cells, and is being used in the study of the histogenesis of radiation-induced bone tumours being carried out in this laboratory. The work presented here is the result of a preliminary investigation of cell division in unirradiated bones by this method. These results for normal bone cells are believed to be of sufficient interest, both as a study of cellular proliferation and as regards the process of endochondral ossification, to warrant separate publication.

Thymidine has been shown to be a specific precursor of D.N.A.* (Reichard and Estborn 1951; Friedkin, Tilson and De Wayne 1956; Amano, Messier and Leblond 1959) and is, with a few possible exceptions (Pelc 1959), taken up only by dividing cells. Furthermore, Hughes (1959) has shown that there is little, if any, turnover of thymidine when once it has been incorporated into the D.N.A. of a cell. Thymidine can be labelled with tritium, a beta-emitting isotope of hydrogen, so that when labelled thymidine is injected into an animal the cells that are in the process of division at that time take up the label and have tritium incorporated into their nuclear D.N.A. The positions of such labelled cells may then be determined by autoradiography of tissue sections. By killing animals at a series of time intervals after injection the progress of these labelled cells may be followed and, even if they divide again, the daughter cells will also be detected on an autoradiograph since the labelled D.N.A. from the parent nucleus will be distributed among them.

Quantitative studies of the uptake of tritiated thymidine into various tissues should be possible if counts are made of the number of photographic grains seen over each cell nucleus, but such studies are complicated by the very short range of tritium beta rays in tissue. The generally accepted values give a maximum range in tissue of 6 to 8 microns and an average range of only about 1 micron (Hughes, Bond, Brecher, Cronkite, Painter, Quastler and Sherman 1958). This short range means that the autoradiographic resolution is excellent (Fig. 11), but also that labelled structures are only effective in producing grains if they lie in the upper 1 or 2 microns of the tissue section next to the emulsion. Thus if grains are seen over a nucleus they are positive proof that it contains labelled D.N.A., but absence of grains does not provide proof of the absence of label because the nucleus may lie at the bottom of the section away from the emulsion.

One further fact that makes tritiated thymidine a valuable tool for the investigation of cellular division is that the labelling process is very rapid. Most of the thymidine is taken up from the blood stream in the first fifteen minutes after injection and the cellular labelling reaches a maximum after one to two hours. This time is short compared with the generation times of most cells (Hughes *et al.* 1958).

METHOD

First generation female hybrids of the August and Marshall strains of rat were used for the study. Both parent strains are highly inbred and the hybrids have been used for tumour experiments because they are more resistant to various diseases than are the pure bred animals.

* The D.N.A. molecule, desoxy-ribonucleic acid, is the basic building unit of the chromosomes in the nucleus. Synthesis of D.N.A. occurs, therefore, whenever the chromosomes are being duplicated prior to cell division.

he rats were 100–120 grammes in weight, that is, six to eight weeks old, when they were elected for experiment.

The standard dose given to each animal was 25 microcuries of tritiated thymidine in 0·5 millilitre of saline at a specific activity of 1·6–1·9 curies/m Mol. Injections were given intravenously or intraperitoneally, work done in this department having shown that there is no sensible difference in uptake between the two methods of injection.

The hind limbs were perfused with formol saline immediately after death and the excised bones were fixed in the same medium. E.D.T.A. (ethylenediamine tetra-acetic acid) neutralised with sodium hydroxide was used for decalcification and it should be noted that nitric acid decalcification was found to produce a loss of label from the cells. The tibia was chosen for study because it has a relatively flat epiphysial cartilage plate at the proximal end. Sections were cut from paraffin wax blocks at 5 microns and floated in pairs on to slides " subbed " with a chrome alum-gelatin solution according to the method of Doinach and Pelc (1950). The sections were covered with Kodak A.R.10 stripping film and exposed for periods of two to six weeks. One set of sections was stained by Feulgen's method before the autoradiographic processing and two sets were stained with Erlich haematoxylin through the gelatin after development of the autoradiographs. The conditions were standardised for all processes, but the slides for any one quantitative study were all developed together in the same solutions.

For the study of dividing cells in the proximal cartilage plate of the tibia a survey was made of 100 columns of cells in the centre of each plate. When there were areas of high background or " lifting " of the section these 100 columns were counted, part on one section, and part on the other section from the same animal. The position of each labelled cell was recorded relative to the other cells in its column; thus a labelled cell at 18/27 was the eighteenth cell up from the metaphysial end of a cartilage column containing twenty-seven cells altogether. The lowest hypertrophied cell with all four walls complete was called cell number 1 in the column and its lower transverse wall defined the " base of the cartilage plate." In these young animals bone formation on the epiphysial side of the plate was not yet complete. Cartilage cells at the top of the columns that appeared to be concerned with epiphysial bone formation were not included in the counts to determine relative cell position in the columns. The grain count over each cell was also recorded.

Labelled cells in the metaphysis at intervals of one hour to seven days after injection were surveyed by scanning a strip of one millimetre width, in bands of 125 microns depth, down through the metaphysis from the base of the cartilage plate (Fig. 1). After seven days the number of labelled cells in the metaphysis was very few, so that the whole width of the bone was surveyed for labelled cells.

In the main experiment two animals were killed at one hour after injection and then one animal at each of the other time intervals from six hours to twenty-eight days. This is a small number of animals for quantitative studies, but the amount of work involved in the counting of grains over 200–300 cells on each section sets a practical limit to the size of the experiment.

Two other experiments were carried out, the first to determine the initial uptake of tritiated thymidine in the cartilage plate at intervals of one-quarter to four hours and in the second experiment two animals were injected every six hours for total periods of one day and three days respectively.

RESULTS

The cartilage plate—Growth curves for the tibia in female hybrid rats of the same ages as those used in the thymidine experiments are given in Figure 2. These curves were derived from radiographic measurements and are given for comparison with the results from the experiments described in this paper. The upper curve shows the overall growth of the tibia, as measured by serial radiographs on four animals. By using data derived by Mrs V. Courtenay

from her experiments in this department (unpublished), the lower curve has been calcula d from the measured values to show that part of the total growth of the tibia which is duc o the division of the proximal cartilage plate.

The series of histograms in Figure 3 give the distributions of labelled cells at the vari us positions in the cartilage plate at intervals of one hour to seven days after injection. Exam es of labelled cells in the cartilage plate are shown in Figures 4 and 5. The first photomicrogr ph shows labelled cells, including one labelled mitosis, at the top of the cartilage plate six h rs after injection and the second shows labelling of degenerate nuclei in the hypertrophied lls at the base of the plate three days after injection. The results in Figure 3 have been corre ed

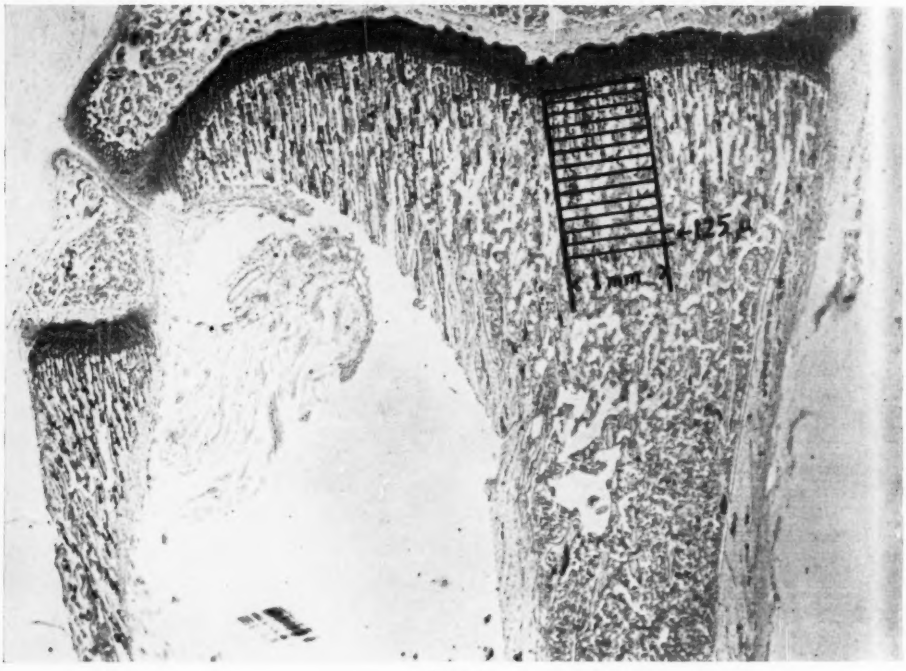


FIG. 1

The proximal epiphysial cartilage plate and metaphysis of the rat tibia. The area in which labelled cells in the metaphysis were surveyed is marked with the scanning bands of 125 micron depth.

to a standard number of cells per column—that is, the average number of twenty-five cells/column. Thus a cell at position 12/20 in a column was adjusted to position 15/25 before being recorded on the histogram. The background count of grains in the emulsion varied from 0·5 to 2·6 grains per nucleus (average 1·2 grains per nucleus), and a minimum of 5 grains per nucleus was taken as the criterion for labelling. These histograms, therefore, depend only upon positive evidence of cell labelling.

From the data relating to the one-hour interval after injection it is clear that few cells divide when they are below position number 12 in the column. Thus positions 12 to 25 define the region of dividing cells. After one day the main region of labelled cells extends down to position number 6, and after two days down to position 2, so that the growth rate appears to be about five cells per day. This number of cells must therefore be lost from the base of the plate each day. Measurements on the hypertrophied cells at the base of the plate show that they have an average height of 25 microns; so the overall growth of the bone due to cell

division in the proximal cartilage plate must be 5×25 microns $= 125$ microns per day. This figure compares well with that of 130 microns per day derived from the radiographic measurements. These results are also comparable with those of Sissons (1955) who obtained a value of $7\frac{1}{2}$ cells per day for the growth rate of the tibia in weanling rats.

If the cells in the fourteen positions from 12 to 25 all divided at the same rate the generation time would be approximately $14/5 = 2{\cdot}8$ days. There is, however, an indication in the one-hour histograms (Fig. 3) that cells in positions 23 to 25 are not labelled as frequently as those in positions 12 to 22, and this would be expected if the generation times of cells in these top three positions were longer than the generation times for the other cells in the dividing region. In order to study this effect more carefully it is necessary to label a larger proportion of the cells and this has been done by giving animals repeated injections at six-hourly

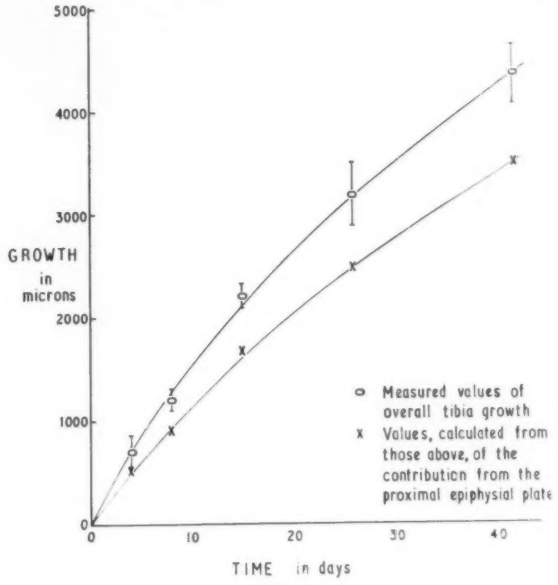


FIG. 2

Growth curves for the overall growth of the tibia and for that part of the growth which is due to cell division at the proximal end. The upper curve is derived from radiographic measurements and the standard deviation of each measurement is shown. The lower curve was calculated from these measured values.

intervals. Provided that thymidine is incorporated into the cells for the synthesis of D.N.A. for a period of longer than six hours in the cell generative cycle, all the cells that are in the process of division while the injections are continued should be labelled with tritiated thymidine. The interval six hours was chosen as it was shorter than the value for the synthetic period derived from preliminary calculations (see later section).

The results for these experiments are shown in Figure 6. Labelled nuclei distribution histograms are given for four and twelve repeated injections, covering total periods of one and three days respectively. Since a 5-micron section is thinner than the width of the cartilage cells not all the cells seen in a section will appear to have nuclei, and for this reason figures for the total numbers of nuclei seen are also included on the histograms in Figure 6. From these results the upper graphs have been calculated to show the percentage of nuclei that have divided in one day and in three days. The only errors that should arise in these graphs are from labelled nuclei that are too deep in the section for their beta rays to reach the emulsion

and produce grains. Examination of the results makes it clear that there must be a variati of generation time between different positions in the columns. After one day of labelli about 60 per cent of the nuclei in positions 16 to 20 are labelled, but this value drops off about a third for the top cell number 25, and this would indicate a generation time of thr times the length of that for the most active cells. After three days of labelling, in positio 13 to 20, over 90 per cent of the nuclei are found to have divided, while less than 50 per ce of the nuclei in position 25 are labelled. The average generation time for cells in the regi of active division must lie between one day, when 60 per cent of them are labelled, and thr days, when practically all of them are labelled. The top four or five cells in the columns ha

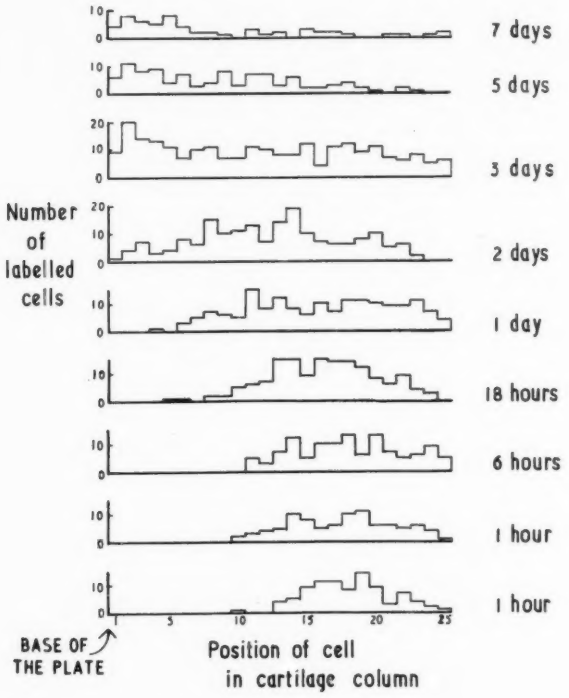


FIG. 3

The distribution of labelled cells in the cartilage plate at intervals of one hour to seven days after injection of tritiated thymidine. The cells are adjusted to twenty-five standard positions within the columns starting with the lowest hypertrophied cell at the base of the plate as number 1.

longer generation times and therefore may be described as forming a zone of reserve cells. Evidently the term "reserve cell" is only relative since the cells in position 25 have a generation time of not more than four times the duration of that for the most active cells. The grain counts over the reserve cells are not obviously lower than the grain counts over the cells in the region of active division. The evidence for the existence of a reserve zone cannot, therefore, be explained away as the result of variation in the geometry of the cells with position in the cell column.

Grain count studies for the cartilage plate—The mean values for the grain counts over cartilage cells are given in the series of histograms in Figure 7. It must be recognised that a range of values is obtained for the grain count in any one position, the values running from zero to

FIG. 4
Labelled chondrocytes near the top of the cartilage plate six hours after injection. One labelled cell in anaphase.

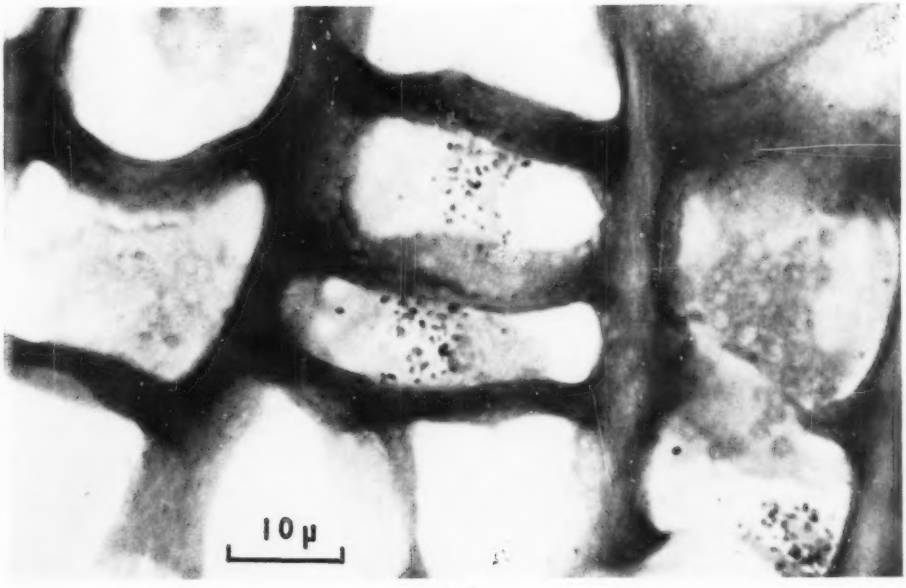


FIG. 5
Degenerate nuclei near the base of the plate, seen labelled three days after injection.

about twice the mean value, for the following reasons: 1) The cell nuclei are at differe t distances below the surface of the section. This effect has already been discussed in t e introduction. 2) The cells will be in different phases of the synthetic period of the cell cyc , at the time of injection. It is not known if the rate of synthesis is constant throughout t s period and, even if it were, a cell that is just completing synthesis at the time of injecti n would take up less thymidine than a cell in the middle of its synthetic period. 3) The triti n atoms disintegrate at a random rate. This variation depends on the total uptake of ea h nucleus and is described by the Poisson distribution law. 4) The cell nuclei vary in size a d shape. A small nucleus will have a relatively larger proportion of its volume within t o microns of the section surface than a large diffuse nucleus. This factor is probably ov r-compensated by the difficulty of counting grains accurately when they are grouped toget er over a small area.

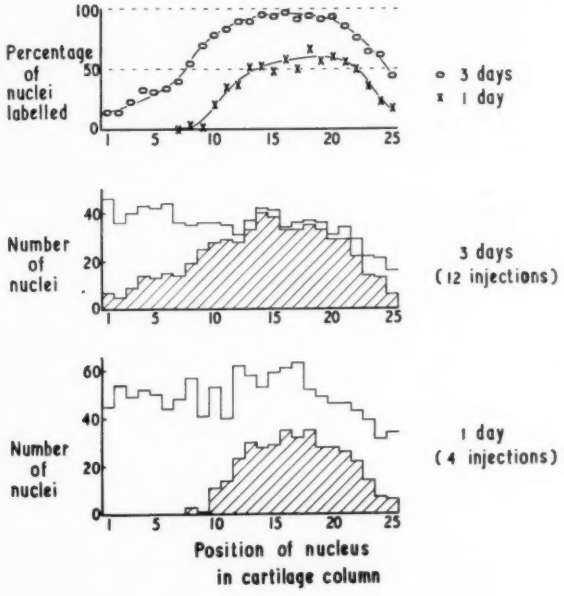


FIG. 6

The two histograms show the distribution of labelled nuclei in the cartilage plate after four and twelve repeated injections given at six-hourly intervals. The shaded histograms show the labelled nuclei and the complete histograms give the total number of nuclei seen. The graphs above show the results plotted as percentages of nuclei labelled.

From Figure 7 it can be seen that, taking any one histogram for the intervals up to one day, there is no significant difference between the mean grain counts over labelled cells in any of the positions in the column. Labelled cells are seen in mitosis at two hours and at six hours, but not at eighteen hours or twenty-four hours; this fact, together with the observation that the mean grain count does not vary significantly with position, would be consistent with the view that all the cells initially labelled had divided once, but only once, before the end of the first day after injection. By two days, however, there is a difference between the average grain counts over cells in the region of division and counts over cells that have passed out of this region. The average for cells in positions 1–5 is thirty-two grains per cell and for cells in positions 12–25 only twenty grains per cell. By three days these two averages are thirty-six for positions 1–5 and fifteen for positions 12–25. Thus, while some of the cells in positions

12–25 must have divided again after one day and before three days, nearly all of them must have completed their second division after labelling before three days, and the mean value for the generation time must lie near two days.

This result depends on comparisons of grain counts over sections from one animal. No conclusions have been drawn from the comparison of grain counts made over sections from different animals because a variation of ±30 per cent was found between the values obtained from the sections in the series from one hour to two days for the total grain counts over all the labelled cells per 100 columns. There should be no significant difference between these values for the total grain count because no labelled cells are lost from the cartilage plate before two days after injection. A correction had already been made to the figures to allow for the variation in the position and the angle of the sections within the bone, and therefore

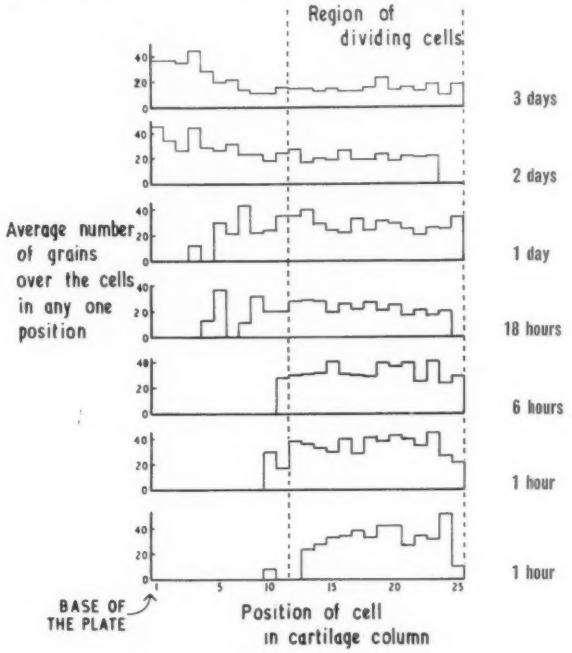


FIG. 7

Values of the average grain count over cells in the various positions down the cartilage plate column at intervals of one hour to three days.

the errors are probably due to a combination of a number of factors, including biological variations from animal to animal, variations in the sensitivity and development of the stripping film and differences in the amount of tritiated thymidine injected.

Lajtha, Oliver and Ellis (1954) divided the generative cycle of the cell into four periods: G_1, the resting period; S, the period of D.N.A. synthesis; G_2, a short resting phase; and M, the duration of mitosis itself. Approximate values for the periods in the mitotic cycle of the cartilage plate cells can be derived from study of these sections.

1) Labelled mitoses are seen in sections from animals killed at two hours after injection, but none has been seen at one hour. Therefore the resting period G_2 must be between one and two hours in duration.

2) The number of mitotic figures seen in an average cartilage plate with 170 columns is 2±4. From the results obtained for Figure 6 it was found that only 50 per cent of the nuclei

are visible in a section, so that the number of mitotic figures should be twenty-four per 70 columns. In each day 170×5 cells will divide and only twenty-four of these are seen to be in mit sis at any one time. Therefore the mitotic time $M = \frac{24 \times 60 \times 24}{850}$ minutes = 41 minutes (approximat y).

3) The synthetic time may be calculated by the same method. In 100 columns ni ty labelled nuclei are seen at one hour. Correcting for the nuclei not seen, this means that 80 nuclei were in synthesis at the time of injection out of a total of 100×5 that will be divi ng during a day. Therefore synthetic time $= \frac{180 \times 24}{500}$ hours $= 8\frac{1}{2}$ hours (approximately).

It should be remembered that these approximations are for the mean values of the per ds for all the dividing cells in the cartilage plate and that the values may vary considerably f m position to position and from cell to cell.

Cell division in the metaphysis—The analysis of labelled cells in the metaphysis is a m re difficult problem. Even in normal sections there are a number of cells which it is difficu to identify. In these autoradiographic preparations with stripping film the process of identifica on is made even harder because the layer of gelatin over the sections prevents the use of m re than one stain, haematoxylin, and because some of the cellular detail is obscured by overly ng photographic grains.

The cells associated with bone, in these sections, have been classified as follows:

1) *Cells with no visible cytoplasm* (with this stain) and large reticular nuclei. These seem to be called by many different names in the literature: primitive connective tissue cells of the marrow by Dodds (1932); haemoblasts and reticulum cells by Jordan (1925); reticulum cells by Barnicot (1941); spindle cells and reticulum cells by Heller, McLean and Bloom (1950). It is perhaps safest to call them mesenchymal cells. In these sections they were seen in the metaphysis, marrow and periosteum. Figure 8 shows unlabelled mesenchymal cells and Figure 9 mesenchymal cells labelled at one hour after injection.

2) *Cells with clearly basophilic cytoplasm and reticular nuclei.* All cells of this type have been classified as osteoblasts, from the pre-osteoblasts of Pritchard (1952), through the typical forms with juxta-nuclear vacuoles, to the flattened cells on the trabecular walls. Labelling in a pre-osteoblast at one hour after injection is shown in Figure 10 and in a more mature osteoblast at three days after injection in Figure 11.

3) *Cells surrounded by osseous tissue or bone*—These are osteocytes and are the simplest to identify. Examples of labelled osteocytes in trabecular bone are shown in Figure 12.

4) *Cells with more than one nucleus*, which varies from a large reticular structure (Fig. 13) to a small densely staining one (Fig. 14). These cells sometimes have a basophilic cytoplasm which envelops the nuclei and is frothy in appearance. These are classified as osteoclasts and normally appear at sites of resorption. The other type of cell most frequently seen to be labelled is the endothelial cell on the wall of a blood vessel.

In Figure 15 histograms are presented to show the distribution of labelling in these four types of cells at intervals of one hour to four weeks after injection of tritiated thymidine. The dynamics of bone growth are seen most clearly if we regard the diaphysis as fixed and the epiphysial plate as growing away from it. Therefore in the histograms in Figure 15 the zero axis of reference for the cell distribution measurements, that is to say the base of the cartilage plate, has been shifted progressively to the right by amounts equal to the epiphysial growth in the corresponding periods (Fig. 2). It can be seen that the labelled mesenchymal cells and osteoblasts migrate up the trabeculae as the base of the cartilage plate regresses, invading the hypertrophied cells one by one as the transverse walls are broken down. Later, when the osteoblasts become embedded in the newly formed bone or become one of the layer of cells lining the trabeculae, they cease to migrate and become fixed in position relative to the diaphysis.

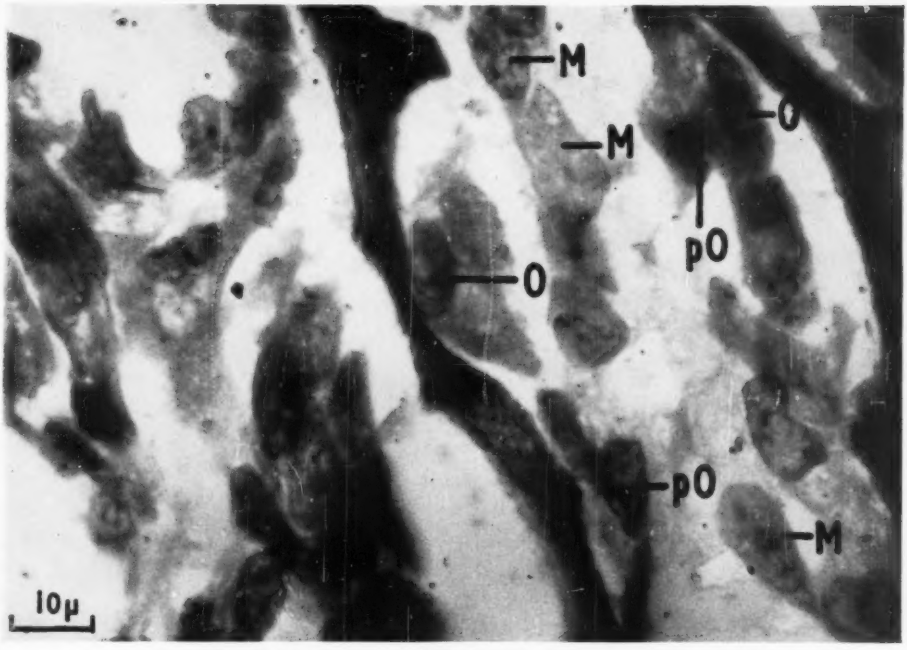


FIG. 8

General view of the types of cell seen in the metaphysis immediately under the base of the cartilage plate. Cells are marked M for mesenchymal and O for osteoblasts (pO for pre-osteoblasts) according to the criteria used in this paper.

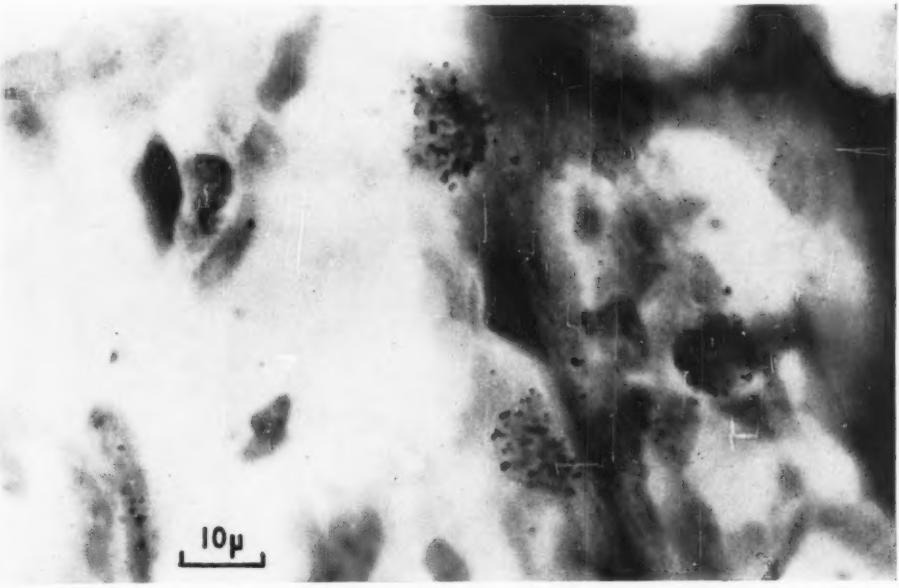


FIG. 9

Labelled mesenchymal cells in the metaphysis. One hour after injection.

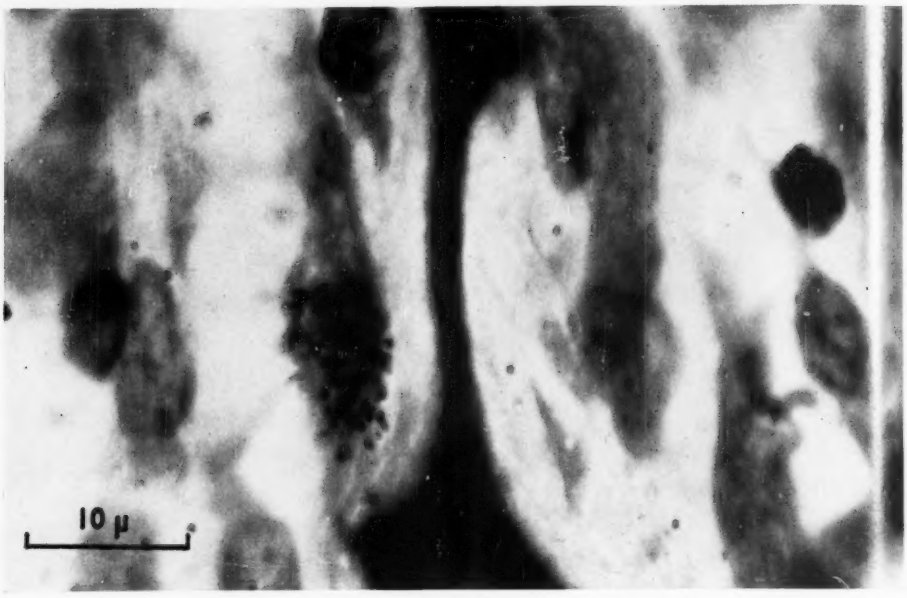


FIG. 10
Pre-osteoblast seen labelled at one hour after injection.

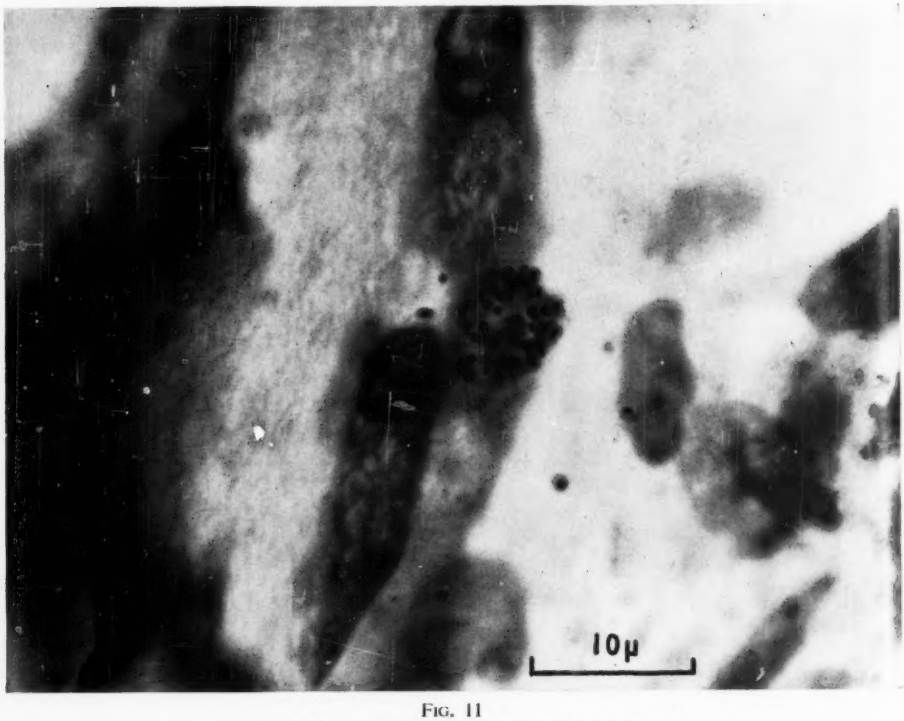


FIG. 11
Labelled osteoblast. Three days after injection.

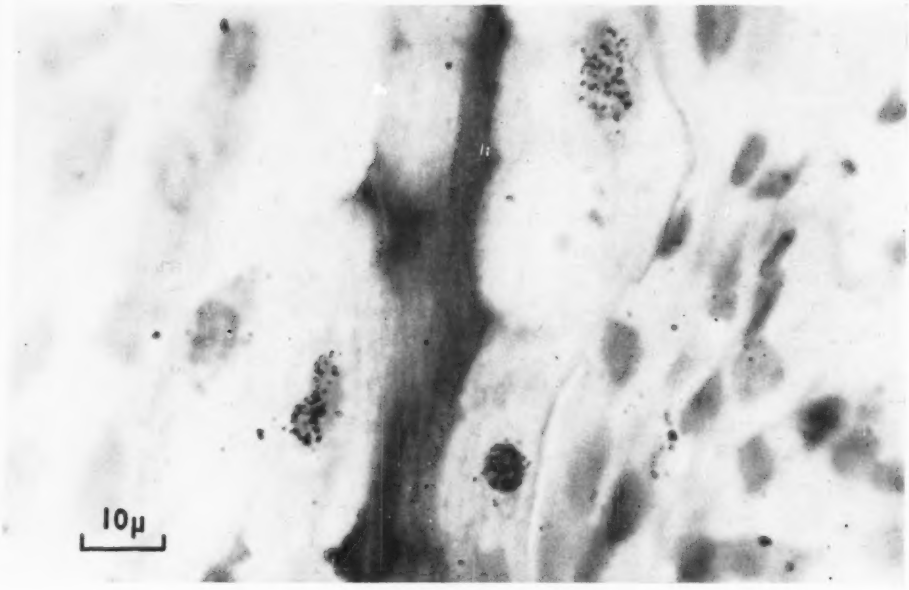


FIG. 12
Labelled osteocytes in the bone laid down on the cartilage bars fourteen days after injection.

Some information about the generation time of these cells can be obtained from the results of repeated injections. These results are presented as the percentage of mesenchymal cells and osteoblasts labelled after one, four and twelve injections (Table I). They were counted in an area 1 millimetre by 0·5 millimetre immediately under the cartilage plate.

These figures indicate that there is a large population of reserve mesenchymal cells that do not divide once in at least three days but which continue to migrate up the trabeculae as

TABLE I
PERCENTAGE OF MESENCHYMAL CELLS AND OSTEOBLASTS LABELLED AFTER REPEATED INJECTIONS

	Cells labelled (*per cent*)	
	Mesenchymal cells	**Osteoblasts**
After 1 injection . .	7	2
After 4 injections in 1 day	32	12
After 12 injections in 3 days	50	26

new bone is formed. The osteoblasts are also fairly slow in turnover, about a quarter of the active population being renewed every three days. Many of these new cells must be replacements of the osteoblasts that become osteocytes or that are left behind as lining cells on the trabeculae.

DISCUSSION

The cartilage plate—This study of the cartilage plate reveals no new basic information on cell division within it. The findings of Dodds (1930) and other workers are confirmed by the observation of mitosis and cellular division occurring in a plane perpendicular to the

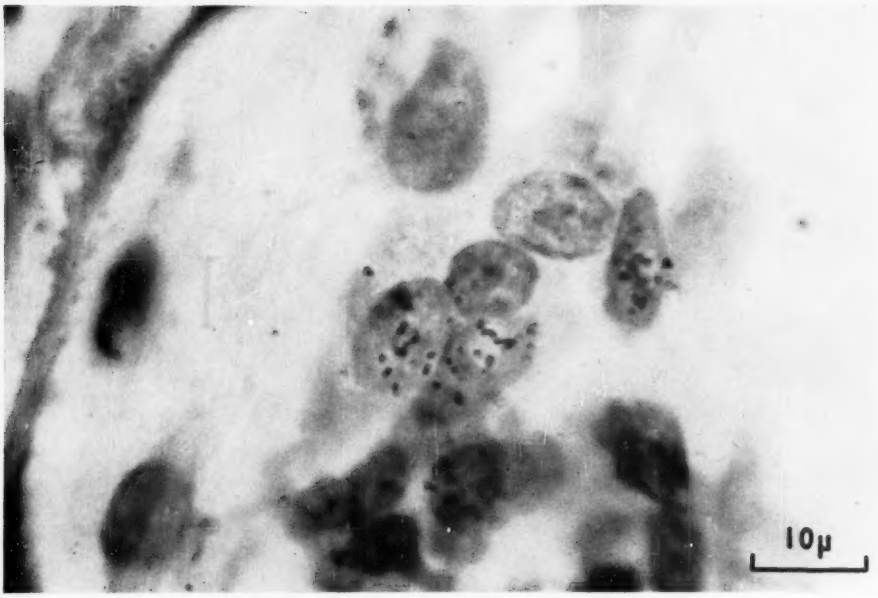


FIG. 13
Osteoclast with two labelled nuclei. Two days after injection. The nuclei are lightly staining and reticular in appearance.

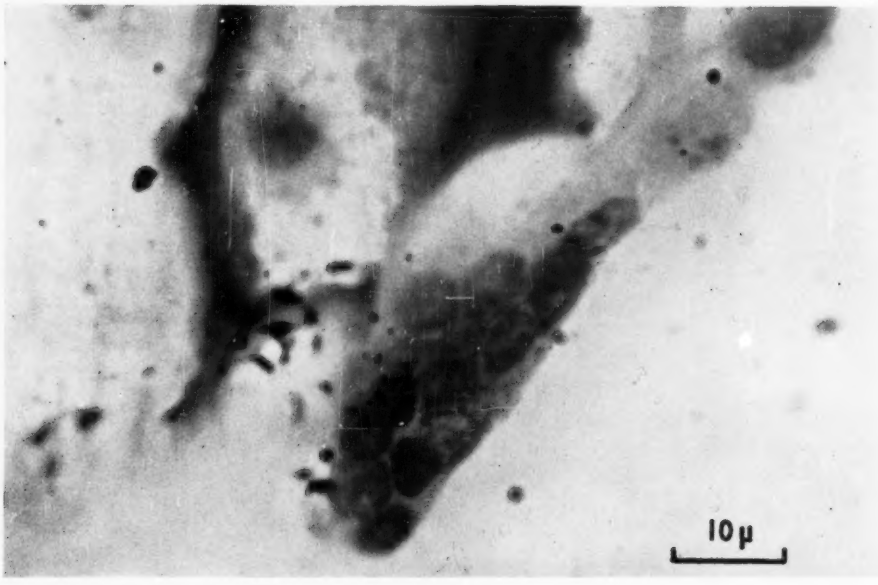


FIG. 14
Osteoclast on the outer surface of the metaphysis three weeks after injection. Small densely staining nuclei with one of them labelled.

direction of growth (Fig. 4). In weanling rats one or two divisions were observed to be taking place in the direction of growth, but such divisions were rare. More precise information is given, however, on the region in which cells do divide, and also some indication of their generation time and mitotic cycle. The presence of reserve cells at the top of the plate is confirmed and at the junction of the articular cartilage with the epiphysial plate one or two well labelled cells were seen three weeks after injection, showing that these cells divide at a very slow rate.

Cell division in the metaphysis—In some studies of endochondral ossification no mention is made of the primitive mesenchymal cells that are found in the region immediately below the

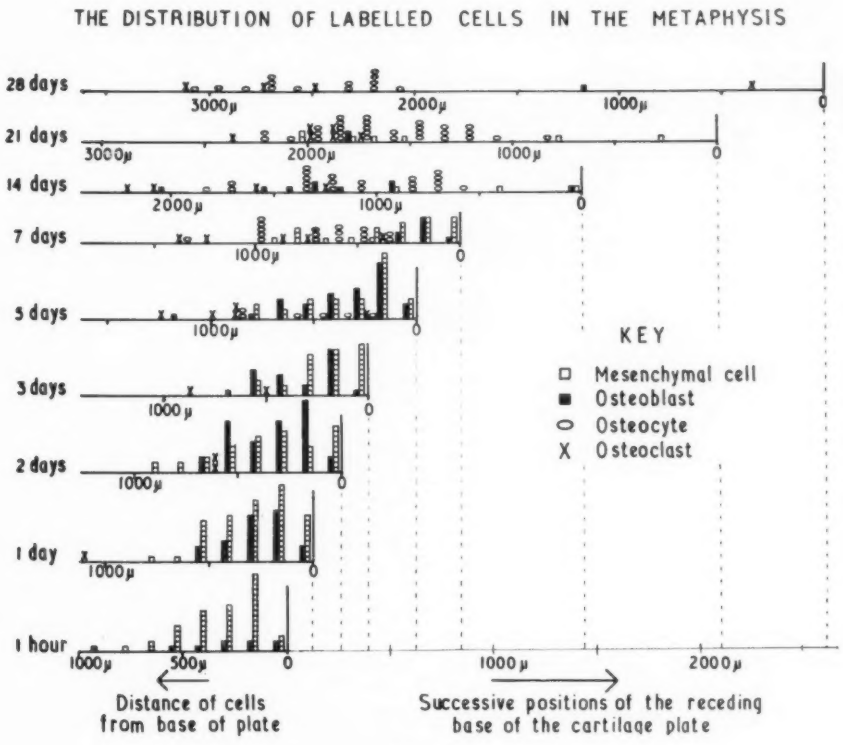


FIG. 15

Distributions of labelling among four types of cells in the metaphysis after intervals of one hour to twenty-eight days. The diaphysis of the bone is regarded as fixed and so the base of the epiphysial cartilage plate has been shifted progressively to the right by distances equal to its growth at the various time intervals shown.

cartilage plate and throughout the rest of the metaphysis. It is these mesenchymal cells, however, that show the highest proportion of tritiated thymidine labelling at early time intervals and must therefore have the highest division rate of all the cells in the metaphysis. They would be expected, then, to play an important part in the process of endochondral ossification, and it is probable that they are the stem cells from which the more specialised cell types are derived.

The importance of the osteoblast and the osteoclast in bone growth has long been recognised, but there is still little sure knowledge about their origin and fate. Recent reviews by Pritchard (1956) and by Hancox (1956) reveal the lack of conclusive evidence about these

two types of cell, and much of the work reported has been studies of these cells under abnorm l conditions. Some further evidence can be drawn from these experiments about the life cyc of these cells under normal conditions.

The osteoblast—Many writers express doubt as to whether osteoblasts ever divide, but it clear from this study that a small percentage do take up thymidine at early intervals (Fig. 1 and it is presumed that they do subsequently divide. Previously mitosis has been the on clue to cell division, and since cells are difficult to identify during mitosis it is understandab that this doubt about the ability of the osteoblast to divide should have arisen. A much larg number of labelled osteoblasts are seen at later time intervals (Fig. 15)—many more, in fac than could be accounted for by the few that do divide. The conclusion must be, therefor that these labelled osteoblasts seen at later time intervals arise by differentiation of other cell The only cells that are labelled in sufficient quantity at earlier time intervals are the mesenchym cells (Fig. 9), and the conclusion that they are the normal precursors of the osteoblast supported by most investigators (Stump 1925; Heller, McLean and Bloom 1950).

The osteoclast—At the earliest time interval of one hour no labelled osteoclast nuclei hav been observed, but a few are found as early as one day after injection. These labelled nucl are large and reticular in form and occur in small osteoclasts. Normally only one of th nuclei in any one osteoclast is seen to be labelled (Figure 13 shows an exception). This fac together with the absence of mitotic figures in osteoclasts, is consistent with the idea tha osteoclasts are able to ingest nuclei from other cells and, in fact, grow by this process. Labelle mesenchymal cells are seen in all regions where there are labelled osteoclast nuclei, and it seems most likely that these mesenchymal cells provide the nuclei for incorporation.

In an osteoclast which has small, dense nuclei, labelling has not been observed before three weeks after injection (Fig. 14), and no evidence was found of the incorporation of osteocytes into osteoclasts.

Migration of cells—One further fact that is emphasised by this study is the migration of large numbers of cells after the retreating cartilage plate at a rate of some 125 microns per day. It is important to keep this migration in mind when considering the effects of bone-seeking isotopes.

SUMMARY

1. The pattern of tritiated thymidine labelling in the cells of the epiphysial cartilage and metaphysis of the tibia in the rat is described for intervals of one hour to twenty-eight days after injection.

2. The region of dividing cells is defined and evidence given for a zone of reserve cells at the top of the cartilage columns.

3. The difficulties of quantitative grain count studies are discussed, and some approximate values are given for the generation time and mitotic cycle periods of the cartilage plate cells.

4. Some further evidence is given about the life cycles of the osteoblast and the osteoclast.

I would like to record my thanks to Dr L. F. Lamerton and to my colleagues at the Institute who have given generously of both advice and help during the various stages of this study. I am grateful to Professor W. V. Mayneord for his interest and encouragement and to Dr H. A. Sissons of the Institute of Orthopaedics for valuable discussions on this work.

I would also like to thank the British electrical industries who financed my Studentship in Biophysics.

REFERENCES

AMANO, M., MESSIER, B., and LEBLOND, C. P. (1959): Specificity of Labelled Thymidine as a Deoxyribonuclei Acid Precursor in Radioautography. *Journal of Histochemistry*, **7,** 153.

BARNICOT, N. A. (1941): Studies on the Factors Involved in Bone Absorption. 1. The Effect of Subcutaneous Transplantation of Bones of the Grey-Lethal House Mouse into Normal Hosts and of Normal Bone into Grey-Lethal Hosts. *American Journal of Anatomy*, **68,** 497.

DODDS, G. S. (1930): Bone Formation and Other Types of Arrangement of Cartilage Cells in Endochondral Ossification. *Anatomical Record*, **46,** 385.

DODDS, G. S. (1932): Osteoclasts and Cartilage Removal. *American Journal of Anatomy*, **50,** 97.

DONIACH, I., and PELC, S. R. (1950): Autoradiographic Technique. *British Journal of Radiology*, **N.S. 23,** 184.

FRIEDKIN, M., TILSON, D., and DE WAYNE, R. (1956): Studies of Deoxyribonucleic Acid Biosynthesis in Embryonic Tissue with Thymidine-C^{14}. *Journal of Biological Chemistry*, **220,** 627.

HANCOX, N. (1956): The Osteoclast. In *The Biochemistry and Physiology of Bone*, p. 213. Edited by G. H. Bourne. New York: Academic Press Inc.

HELLER, M., MCLEAN, F. C., and BLOOM, W. (1950): Cellular Transformations in Mammalian Bones Induced by Parathyroid Extract. *American Journal of Anatomy*, **87,** 315.

HUGHES, W. L., BOND, V. P., BRECHER, G., CRONKITE, E. P., PAINTER, R. B., QUASTLER, H., and SHERMAN, F. G. (1958): Cellular Proliferation in the Mouse as Revealed by Autoradiography with Tritiated Thymidine. *Proceedings of the National Academy of Sciences*, **44,** 476.

HUGHES, W. L. (1959): The Metabolic Stability of Deoxyribonucleic Acid. In *Kinetics of Cellular Proliferation*. p. 83. Edited by F. Stohlman, Jun. New York and London: Grune & Stratton.

JORDAN, H. E. (1925): The Experimental Production of Osteoclasts in the Frog, *Rana pipiens*. *Anatomical Record*, **30,** 107.

LAJTHA, L. G., OLIVER, R., and ELLIS, F. (1954): Incorporation of ^{32}P and Adenine ^{14}C into DNA by Human Bone Marrow Cells *in vitro*. *British Journal of Cancer*, **8,** 367.

PELC, S. R. (1959): The Metabolic Activity of DNA as Shown by Autoradiographs. *Laboratory Investigation*, **8,** 225.

PRITCHARD, J. J. (1952): A Cytological and Histochemical Study of Bone and Cartilage Formation in the Rat. *Journal of Anatomy*, **86,** 259.

PRITCHARD, J. J. (1956): The Osteoblast. In *The Biochemistry and Physiology of Bone*, p. 179. Edited by G. H. Bourne. New York: Academic Press Inc.

REICHARD, P., and ESTBORN, B. (1951): The Utilization of Desoxyribosides in the Synthesis of Polynucleotides. *Journal of Biological Chemistry*, **188,** 839.

SISSONS, H. A. (1955): Experimental Study on the Effect of Local Irradiation on Bone Growth. In *Progress in Radiobiology. Proceedings of the Fourth International Conference on Radiobiology*, p. 436. Edited by J. S. Mitchell, B. E. Holmes and C. L. Smith. Edinburgh and London: Oliver & Boyd.

STUMP, C. W. (1925): The Histogenesis of Bone. *Journal of Anatomy*, **59,** 136.

MUSEUM PAGES

V. The Sesamoids of the Hallux

CAMPBELL GOLDING, LONDON, ENGLAND

It often seems to the radiologist in an orthopaedic hospital that radiographs of the fee outnumber all others, and that it is his stultifying duty to write reports on a region that seldon presents any feature of interest.

In recent years comparatively little has been recorded on pathological changes in th sesamoid bones of the hallux, partly because it is a subject of small practical importanc with limited scope for original observation, and possibly because the historical, embryological histological and anatomical aspects have been described in great detail in the past. It woul probably be a surprise to any student to find that in 1936 a monograph of 108 pages wit ninety-seven references was written on the sesamoids of the hallux (Kewenter), and i 1949 a radiological study was made (Hubay) on the hallux in 510 patients. The literatur before 1930, however, was clearly full of inaccurate observations and false conclusions, largel because of the difficulties in the interpretation of radiographs. These difficulties were no appreciated at that time. Some are illustrated in this paper.

DIFFERENTIATION BETWEEN FRACTURE AND CONGENITAL VARIATION

The diagnosis of a *multipartite* variation is never in doubt. A *bipartite* sesamoid may b obviously a congenital abnormality, but in some cases the differentiation from fracture i difficult, and it cannot always be made from routine observations.

There are some cases in the series under consideration which suggest that stress fracture of the sesamoid bones may not be uncommon, and may remain ununited.

In one case the patient was a surgeon who felt a sudden pain under the first toe while standing in the operation theatre. The radiograph showed a divided medial sesamoid with sharp edges. This was believed to be a fracture; it was ignored with success (Murray 1960).

In another case a young woman complained of pain in the metatarso-phalangeal joint of the hallux, worse at night and increased by flexion or stretching of the toe. There was no history of injury. Radiographs showed a division of the medial sesamoid bone into two parts, with sharp edges (Fig. 1). The opposing sesamoid was intact. This condition might b interpreted either as a fractured or a bipartite medial sesamoid. She was treated for eighteen months with varying success but with ultimate relief. She returned six months later with a recurrence of pain, and a radiograph demonstrated that the two halves of the bone were then widely separated (Fig. 2). There was no history of an injury at any time.

A similar condition occurred in another woman, aged eighteen, who had an early bunion on the left side. An antero-posterior radiograph was done, which coincidentally showed th right foot. No abnormality was seen on the left side, but the medial sesamoid on the right was in two parts (Fig. 3). There had not been any history of injury on this side and there wer no symptoms. Two months later the patient returned, having fallen and injured the right first toe. A radiograph demonstrated that the two fragments were now widely separated. The symptoms subsided rapidly and no operation was necessary (Fig. 4).

It is interesting to speculate on the factor which caused the original radiographi appearances in the last two cases described above. Ordinary fracture can be excluded becaus there was no history of injury. Were they congenitally bipartite bones? This is improbabl

FIG. 1 FIG. 2 FIG. 3 FIG. 4

Figure 1—There is a narrow gap in the medial sesamoid. Figure 2—Two years later, there is a wide gap in the sesamoid. No history of trauma. Figure 3—The appearance is similar to Figure 1. There were no symptoms in the foot. Both feet were radiographed together. The symptoms were on the opposite side. Figure 4—After trauma, there is separation of the two parts of the sesamoid.

FIG. 5 FIG. 6

Figure 5—The sesamoids appear to be normal. Figure 6—There is osteochondritis of the medial sesamoid (same patient as Figure 5).

FIG. 7
Osteochondritis of the lateral sesamoid with all symptoms referred to the medial side.

because in that condition separation would be unlikely t occur. It seems likely that they were stress fractures. recently reported stress fracture of the patella (Devas 196 bears some resemblance to these lesions.

OSTEOCHONDRITIS OF SESAMOID BONES

In this condition, first described by Renander (1924 the radiological appearances are like those of oste chondritis of the lunate bone. It is the medial sesamoi which is usually involved by osteochondritis and b fracture, and the differentiation between them canno always be made in antero-posterior or routine obliqu views, in which the sesamoids are obscured by th metatarsals. The patient shown in Figure 5 had pain i the joint with tender sesamoids. The antero-posterio radiograph did not show a definite lesion, but oste chondritis of the medial sesamoid bone was clearl demonstrated in an axial projection (Fig. 6). Thre months after treatment she was free from symptoms.

Any clinician who is familiar with the lesions of Schlatter's disease or osteochondritis o the apophysis of the calcaneum must have found some difficulty in relating the radiological signs to the patient's complaint. This discrepancy is clearly shown also in the sesamoid bones. The patient shown in Figure 7 referred all her symptoms accurately to the medial sesamoid bone, which was consistently tender. She had had pain for two years, aggravated by standing and walking and relieved by rest. She also developed pain in the calf and back of the thigh.

FIG. 8

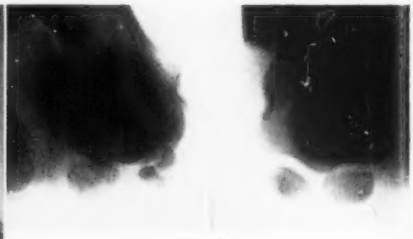


FIG. 9

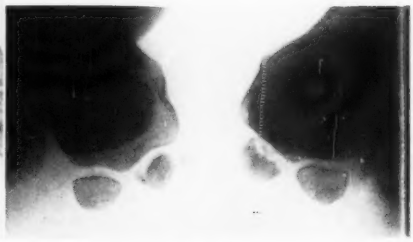


FIG. 10

Figure 8—The appearance of the sesamoids is within normal limits. Figure 9—The axial film show osteochondritis of the medial sesamoid on the right. Figure 10—Three years later: the appearance of the righ medial sesamoid has improved; but the left medial sesamoid has begun to disintegrate, without local symptoms.

which she associated with the pain in the big toe, and she had occasional swelling of the inner side of the foot. She has been under observation now for a year. The surgeon expressed the opinion that, if any surgery was required, it would be for the medial sesamoid, and he was not prepared to do anything until the fragmentation of the lateral sesamoid was stationary.

The patient whose film is shown in Figure 8 was a woman of nineteen who complained of pain in the right metatarso-phalangeal joint of the first toe. It started in the middle of the night while she was on holiday. There was no history of injury. She could not walk without limping. The pain diminished, but she was unable to wear anything but soft low-heeled shoes. The radiograph shows old osteochondritis of the second left metatarsal bone, which was symptomless, and a minor abnormality of the right medial sesamoid. This might pass without

FIG. 11 FIG. 12

Figure 11—The break in the medial sesamoid may be interpreted as a fracture or congenital anomaly. Figure 12—The tilted lateral film demonstrates, however, that there is osteochondritis.

comment, but an axial film demonstrated that this was due to osteochondritis (Fig. 9). She attended hospital for six months and was not seen again for two and a half years, when she returned because of a recurrence of pain in the first right metatarso-phalangeal joint. At that time the appearance of the medial sesamoid bone had changed considerably (Fig. 10) and she was admitted for removal of the bone. It was also noticed that the medial sesamoid bone of the left toe had begun to disintegrate but was symptomless.

Apart from the confusing history that the patient may give, the radiological problem is simple enough if adequate examination is made. This is illustrated by Figure 11. The patient was a radiographer who complained of pain in the first metatarso-phalangeal joints of both feet. She still has pain on the side where there are no radiological changes, whereas the pain in the joint which is illustrated has subsided. This abnormality might be interpreted as a fracture or a bipartite bone; it looks rather more like the former, but the oblique film (Fig. 12) shows that it is osteochondritis.

REFERENCES

DEVAS, M. B. (1960): Stress Fractures of the Patella. *Journal of Bone and Joint Surgery*, **42-B**, 71.

HUBAY, C. A. (1949): Sesamoid Bones of the Hands and Feet. *American Journal of Roentgenology*, **61**, 493.

KEWENTER, Y. (1936): Die Sesambeine des 1. Metatarsophalangealgelenks des Menschen. *Acta Orthopaedica Scandinavica*, Supplementum 2.

MURRAY, R. O. (1960): *Personal communication.*

RENANDER, A. (1924): Two Cases of Typical Osteochondropathy of the Medial Sesamoid Bone of the First Metatarsal. *Acta Radiologica*, **3**, 521.

Douglas Glass

ARCHIBALD HECTOR McINDOE
1900–1960

Archibald McIndoe died peacefully in his sleep from a coronary occlusion on April 12, 1960, at the age of fifty-nine.

McIndoe was born in New Zealand, qualified in medicine at the University of Otago, and did his postgraduate training in surgery at the Mayo Clinic, for which for the remainder of his life he had the greatest affection and admiration. Later he came to London and joined his cousin, Sir Harold Gillies, the great pioneer of plastic surgery, who outlived him by a few months. Within a short time he was on the staff of St Bartholomew's Hospital and his future in London was secure; indeed, for the last twenty years of his life, he was probably the most successful surgeon in any speciality in the metropolis.

He was a great friend of orthopaedic surgery. During the second world war he was consultant in plastic surgery to the Royal Air Force. The writer became closely associated with him in the problems presented by burns combined with fractures, and in the management of patients with extensive skin and bone loss. This work, which started in Royal Air Force hospitals and at the Queen Victoria Hospital, East Grinstead, was continued at the latter hospital until his death.

On his election to the Council of the Royal College of Surgeons of England he became intensely interested in the College, of which he had just ceased to be Senior Vice-President when he died. There was little doubt that he would have been the next President and the first New Zealander to hold the highest order in British surgery. His loss is a sore one for, among his many qualities, was an outstanding ability to persuade the giants of industry and commerce to contribute to the rebuilding and upkeep of the College.

Archie, as he was affectionately known to all his friends, was a great plastic surgeon and a great teacher. But he was also the most likeable of men, with an infinite capacity for enjoying life in the company of every stratum of society. Honours were given to him in abundance but, though accepted with obvious delight, they never altered his delightful character. He will be greatly missed by his many friends and colleagues all over the world—and not least by his patients, especially the badly burnt Royal Air Force boys of the second world war, banded together to form the Guinea Pig Club which met annually at East Grinstead under his presidency. To his closest personal friends his loss is irreparable. H. O.-C.

GORDON GORDON-TAYLOR
1878–1960

There are few men of whom another can say " I love him," but G.-T. was such a one—surely he was the best-loved surgeon in the world. Those who were privileged to serve him came under his spell at once, and when their apprenticeship was over they found they had gained a life-long friend. Age is a factor he hardly recognised, unless it was that he sought the company of the young rather than the old. He talked to his house-surgeon like a colleague and he was so vivid a raconteur that nearly as much surgery could be learned whilst sitting with him in his car journeying around the country as from watching him at work. It is often held that apprenticeship to a master craftsman is the best way of learning; the fortunate men who received even part of their training from him will testify to the truth of this dictum. He managed somehow to instil into his pupils the very essence of the spirit of surgery and not one of them can ever forget the lesson.

Gordon-Taylor was almost the last survivor of a great generation of surgeons, the pioneers of twentieth-century surgery, and he was amongst the greatest of them. These men began their careers at the time asepsis was replacing antisepsis, and advances in anaesthesis were making longer operations comparatively safe. He played a large part in the development of abdominal surgery, particularly of the stomach, and in the radical treatment of cancer at all sites—he had no use for half measures when dealing with malignant disease. Essentially a clinician, his judgment was clinical and he had a profound distrust of the statistical presentation of surgical results. His memory of patients was

magnificent, and he much preferred to recall the result of treatment of an identical condition in Mrs X than the average result in a hundred more or less comparable lesions. And when, twelve years ago, he himself was found to have a carcinoma of the transverse colon his first thought was of the particular people of similar age on whom he had operated: most had survived for many years and he was much comforted.

Although a child of the days when the general surgeon ruled supreme he was a firm believer in specialism. It is probably true to say that he regarded himself as a surgeon specialising in the treatment of cancer and of surgical conditions of the abdomen. Certainly he supported the development of specialities when he thought they could make a contribution, but he strongly opposed those he regarded as " phoney." He was a good friend of orthopaedics and a contributor to this Journal.

G.-T. lived for his patients and for his surgery. His patients adored him; the famous red carnation was delivered early each morning for many years by a florist on whose wife he had operated, and later it was the daily gift of another patient. He could not understand how surgeons could live in the suburbs or country, thus making it difficult to visit in the late evening the patients they had operated upon that day. He had few interests outside surgery, which he thought was a whole-time job for any man who took it seriously, and when his active days were passed he devoted himself to the welfare of the rising generation of surgeons of the whole Commonwealth. It was characteristic that, when visiting another country, he preferred to live in the residents' mess rather than accept the hospitality of one of the senior surgeons. His hobbies were walking, dancing and watching cricket—it was when leaving Lord's Cricket Ground that he met his tragic end. He never owned a car and walked around London whenever possible (when in active practice he had a Rolls-Royce on permanent hire, always with the same driver). And his idea of a holiday was to take the Saturday night train to Scotland, walk across the Cairngorms on Sunday, and spend Monday in Edinburgh watching Wilkie at work before returning on the night train.

A great man has died and all who knew him mourn him. P. W.

PROCEEDINGS AND REPORTS OF UNIVERSITIES COLLEGES, COUNCILS AND ASSOCIATIONS

GREAT BRITAIN AND IRELAND

VISIT OF THE 1960 TRAVELLING FELLOWS TO THE UNITED STATES OF AMERICA AND CANADA

Among the passengers on the *Queen Elizabeth* when she sailed from Southampton on April 7, 1960, were four Travelling Fellows of the British Orthopaedic Association: J. W. Dickson (Ipswich), A. H. C. Ratliff (Southend), R. H. C. Robins (Exeter) and J. H. S. Scott (Edinburgh). W. G. Doig (Melbourne) joined the party at New York.

On arrival, we were met by Dr Edward Winant and our passage through the customs was facilitated by a grateful orthopaedic patient who happened to be a senior official of the U.S. Customs and Excise. After a financially necessary visit to Wall Street, the remainder of the day was given to sightseeing before we assembled at the Columbia University Club for a welcoming dinner, attended by a most distinguished gathering of American orthopaedic surgeons, with Dr Philip D. Wilson in the chair.

Left to right—A. H. C. Ratliff, J. W. Dickson, W. G. Doig, R. H. C. Robins, J. H. S. Scott at Portland, Oregon.

On this occasion the tour had a distinct western and southern flavour; first we went to the middle-west and then to western Canada; from there down the north Pacific coast, through California to Texas, and then back through the southern states to Hot Springs, Virginia, for the meeting of the American Orthopaedic Association. A few days in Washington and New York completed the tour. It was the longest tour planned for the Travelling Fellows, and the first visit to Texas.

Throughout the whole tour we received wonderful and generous hospitality and no evening passed without the most warm-hearted entertainment at which we enlarged our acquaintance by meeting the families and friends of our hosts. It is unfortunate that space does not permit us to mention all our hosts and hostesses by name or to describe their beautiful homes. We were also glad to be able to understand the American system of training, and on our part we tried to explain the British system and to correct misconceptions about the National Health Service in which very great interest was shown.

We were impressed with the wide variety of research projects, including the great amount of animal work, and the importance attached to detailed bone pathology. We had expected to see much new building and we were not disappointed. The work devoted to protheses for the amputee was

very evident. We saw great achievements in rehabilitation utilising everything from enthusiasm to electronics, and we were impressed by the high standards at the Shriners' Hospitals for crippled children.

In the clinical field the subjects which were repeatedly discussed in different forms were scoliosis, fractures of the neck of femur, the use of intramedullary fixation for such varied conditions as fractures of the forearm and osteogenesis imperfecta, the surgery of cerebral palsy and the rheumatoid hand, and regional perfusion with chemotherapeutic agents for malignant disease.

Ann Arbor (*April* 13–15)—We were guests of Dr Cari Badgley and Dr Robert Bailey and we stayed in the Union of the University of Michigan. After flying in to Detroit at mid-day we attended a scientific session in the University demonstrating the work of the Department of Surgery, including a paper on central cord compression. At dinner we were honoured by the presence of Dr Fred Coller in the chair. The following morning was devoted to operating sessions, and Dr Badgley showed his method of cup arthroplasty. In the afternoon we visited the Ford works and the Henry Ford museum, Detroit, by courtesy of Dr Leslie Mitchell and dined at his home afterwards. A full and varied clinical programme on the last morning included instances of the fibro-elastic diathesis and some excellent results of cup arthroplasty.

Chicago (*April* 16–20)—Dr Edward Compere arranged our visit and, with Dr William Schnute, Dr Harold Sofield and Dr John Fahey, entertained us as house guests. The first day was spent at the University of Illinois, where Dr Robert Ray presented a series of rare cases before showing us his research laboratory where we were impressed by the wide variety of projects in hand, including studies of bone growth in tissue culture.

Easter was spent at the country home of Dr Schnute on Beverley Shores, Indiana, and we visited the great Lutheran Church at Valparaiso. This week-end provided the only cold weather during the whole tour.

Dr Compere was chairman of the programme at the hospitals associated with Northwestern University, which included a long-term follow-up of 175 radium dial workers with radionecrosis by Dr Finkel, and a clear account of orthopaedic training by Dr Sam Banks. Dr Lambert demonstrated recent advances in prosthetics for the amputee and the techniques of design and construction at the Rehabilitation Institute.

At the University of Chicago, with Dr Robert Moore in the chair, we participated in a conference on fractures with undergraduate students who appreciated our tendency to disagree amongst ourselves.

A very enjoyable morning was spent at the Shriners' Hospital, where Dr Sofield demonstrated his technique of multiple osteotomy for osteogenesis imperfecta.

With Dr Fahey at St Francis's Hospital, Evanston, the clinical discussion ranged over xanthomata, rib tumours, bone cysts, dislocations of the talus and slipped upper femoral epiphyses—with demonstration of remodelling after pin fixation in a displaced position. A long follow-up of femoral neck fractures with annual domiciliary radiographs was particularly impressive.

Rochester (*April* 21–24)—The first impression of the Mayo clinic is one of a fabulous organisation, and further acquaintance shows that this efficiency extends to all its functions. We were guests of the orthopaedic staff and on the first day were entertained to lunch at Mayo Foundation House, which is now a memorial to the two Mayo brothers. The programme consisted of scientific sessions, tours of the clinic and operating sessions: Dr Herman Young tackled a very difficult ununited femur; Dr William Bickel discussed the surgery of comminuted elbow fractures; Dr Paul Lipscomb reported the results of arthrodesis of the hip and demonstrated the separation of a syndactyly of the hand; Dr Edward Henderson discussed the problem of fixed pelvic tilting; Dr J. C. Ivins presented aspects of vascular surgery; and Dr A. J. Bianco that of intervertebral disc infection in children. Among the scientific presentations was an excellent film on bone tumours made by Dr Mark Coventry and Dr D. C. Dahlin.

The entertainment included a progressive dinner at the country homes of Dr Coventry, Dr Lipscomb and Dr Young on "Bony Ridge," barbecue steaks and a display of the aurora borealis at Dr Bickel's home, and an attempt at water skiing with Dr Henderson which emphasised the need for well fitting bathing trunks.

Minneapolis (*April* 25–26)—We were house guests of Dr John Moe and Dr Roland Neumann, and in a short but full programme we visited the Gillette Children's Hospital, University of Minnesota and the Veterans' Administration. The accent was on scoliosis, including a film on operative technique, a discussion of respiratory problems and the treatment of pseudarthrosis. The low incidence of pseudarthrosis in Dr Moe's series is a tribute to his technique of articular facet fusion. Among other papers of great interest were those on diastematomyelia by Dr Sheldon Lagaard and osteotomy of the pelvis for ectopia vesicae by Dr Harvey O'Phelan.

Edmonton, Calgary and Banff (*April* 27–30)—After a long journey by air we were met by Dr Gordon Cameron and Dr Olav Rostrup, representing the orthopaedic surgeons of Edmonton who were our hosts. The morning programme was introduced by Dean Walter C. MacKenzie, Professor of Surgery, and moderated by Dr Rostrup. A technique of limb perfusion was shown and a series of patients with fat embolism discussed. In the sphere of rehabilitation examples of the work of the upper limb reconstruction unit were presented, followed by the Canadian Limb for hip disarticulation. The layout of the physiotherapy department was noteworthy and the McEachern department of surgical research was one example of the great facilities available for work of this kind in North America.

We spent a fascinating afternoon with Dr Gordon Gray at the Charles Camsell Hospital, serving the Eskimo and Indian population for 1½ million square miles of the N.W. territories. Tuberculosis remains a serious problem here; some of the home-made prostheses devised by Eskimo patients were an interesting contrast to what we had seen that morning.

The following day we were driven to Calgary by Dr Rostrup and Dr Graham Huckell, and we remained their guests during our stay in Banff. The afternoon programme at the children's hospital was notable for a report on patients with fibrosarcoma treated by local resection. Dr Gordon Townsend presided and entertained us to dinner at his home before we were driven to Banff in the dark.

We awoke next day to the superb view of the snow-capped Rockies bathed in brilliant sunshine and arose to find a bear in the dustbin outside the back door. The experiences of this unforgettable day included a drive to Lake Louise, still frozen over, a perilous trip up Mount Norquay by ski-lift and subsequent thawing in hot sulphur springs. On the next day we boarded the transcontinental train of the Canadian Pacific Railway for an incomparable journey through the mountains.

Vancouver (*May* 1–4)—Leaving the wintry landscape of Alberta we awoke in the full flood of the British Columbian spring, with Vancouver ablaze with azaleas. We were house guests of Dr H. H. Boucher, Dr C. Cook, Dr F. Preston, Dr Cameron Allen and Dr E. C. H. Lehmann. During our stay visits were made to Shaugnessy Hospital, the Rehabilitation Centre and the University of British Columbia. Dr Boucher spoke on his method of spinal fusion, which employs screw fixation across the facets as an adjunct to grafting, and demonstrated the surgical technique. Dr J. C. Colbeck gave an account of a detailed study of hospital infection, emphasising the fallacy of moist drapes and gowns as a bacteriological barrier. Amongst other presentations Dr Frank Patterson spoke on sciatic nerve injuries, Dr H. S. Gillespie on partial and total excision of the scaphoid bone and Dr C. S. Allen on injuries of the forefoot; Dr Donald Starr gave a spirited review of Brittain's arthrodesis of the hip, and Dr A. C. McConkey described the use of the Ken nail for fractures of the femoral neck.

Seattle (*May* 5–6)—On arrival by train we were greeted by Dr John le Cocq, and by Dr G. Truell and Dr E. Burgess, with whom we stayed at their beautiful lakeside homes. Unfortunately the weather was unsettled and we did not see the lake at its best.

At the University of Washington, where Dr D. K. Clawson has succeeded Dr Ray as head of the Division of Orthopaedic Surgery, we were shown the research work in orthopaedic surgery, which included observations on the fixing of tetracycline antibiotics in bone and Dr Kirk Anderson's work on bone implants. We also saw some beautiful stereoscopic photographs in colour of anatomical specimens prepared by Dr D. Bassett. At the Swedish Hospital Dr Le Cocq presented an interesting series of patients with hip operations observed for many years and Dr Roger Anderson demonstrated the versatility of his method of fixation of fractures.

A morning at the Children's Orthopaedic Hospital included demonstrations by Dr N. Duncan on reflex activity in cerebral palsy and by Dr Burgess on amputations. After a dinner given by the Puget Sound Chapter of the Western Orthopedic Club we were shown a most interesting and historic film on the Clinic System and papers were read by the Travelling Fellows.

Portland (*May* 7–9)—We arrived at the week-end and were driven by Dr Joe Davis and Dr L. Noall to a country house on the rocky north Pacific coast, where we were guests of Dr and Mrs William Snell. There we enjoyed a day of relaxation in the loveliest scenery.

The clinical programme at Portland was divided between the Shriners' Hospital and the University of Oregon. At the former there were clinical demonstrations by Dr Leo Lucas and by Dr E. C. Chuinard of osteotomy of the calcaneum for relapsed club foot and of the femur for congenital dislocation of the hip. In each case we entered the operating room at the critical stage of the procedure. The use of osteotomy was also demonstrated in the treatment of deformity in Erb's palsy, incorrectable equinus of the foot, and for internal torsion of the tibia. Dr Snell reported a series of patients with monarticular non-specific arthritis.

At both Seattle and Portland we were introduced to the public on television. At the University Dr Joe Davis described his technique of pedicle bone grafting in arthrodesis of the hip and shoulder and Dr F. Fitch reported an interesting series of knee fusions.

San Francisco (*May* 10)—Through the courtesy of our hosts at Portland we spent a delightful d
exploring this beautiful city.

Los Angeles (*May* 11–13)—The size of this city was impressive as we flew in at night. We started
Beverly Hills, where we were entertained by Dr Paul McMaster and Dr Robert Mazet. At t
clinical conference at the Veterans' Administration, Dr McMaster discussed vertebral osteoto
for ankylosing spondylitis and Dr Mazet showed cases of delayed union of a fractured scaphoid bo
which had united with prolonged immobilisation after an interval of as much as a year between t
injury and commencement of treatment. Dr Louis Lichtenstein presented some deceptive cases
bone pathology and Dr Marshall Urist described the uses of collapatite and his method of arthropla
of the hip. In the afternoon we visited the studios of 20th Century Fox Films and viewed Mr Bi
Crosby in female garb.

Route of the Travelling Fellows 1960.

On the following morning Dr A. Neufeld took the chair at a clinical conference at the Gener
Hospital of the University of Southern California.

In the afternoon we visited the beautiful Los Angeles Orthopaedic Hospital, one of the mos
modern in the United States. We were honoured by the presence of Dr Charles Lowman who, with
Dr Vernon Luck, described the use of the fascial transplant operation which bears his name. Dr Luck
also gave his views on the etiology and treatment of Dupuytren's contracture. Dr Joseph Boyes
showed a beautiful series of tendon transfers in the hand for peripheral nerve injury. Dr Joseph
Risser described the localiser cast for correction of scoliosis and demonstrated its application.

On our last day we were privileged to be introduced by Dr Vernon Thompson to the work of th
Rancho los Amigos, a hospital which caters for those severely disabled by disorders of the respirato
and locomotor systems, and which in the five years from 1952 treated 2,598 patients with poliomyeliti
half of them respiratory. We were at once struck by the atmosphere of enthusiasm in both patien
and staff. Dr Vernon Nickel and his associates, Dr Jacqueline Perry and Dr Alice L. Garrett,
demonstrated to us the use of the halo splint for control of the paralysed neck and the possibiliti
offered by total spinal fusion in certain cases of extensive paralysis. Methods of improving respiratio
were described and the surgical reconstruction of the upper limb was discussed in relation to function
bracing; the flexor hinge splint was demonstrated and also the use of the McKibben artificial muscl

A research department is now devoted to the development of the latter. Before we had time to take breath the hospital staff swept us away to the fantasies of Disneyland and the Wild West of Knott's Berry Farm.

San Antonio (*May* 14–18)—Our hosts were Colonel Milton Thompson, Dr John Hinchey, Dr Ray Davis, Dr Philip Day and Dr David Oliver. We flew into a temperature of 96 degrees and were grateful for the cool comfort of the oldest hotel in the south-west. During the week-end we visited many places of interest including the old Governor's Palace, the San Jose Mission and the Alamo. The last three are fine buildings dating from the early Spanish colonial period; we noted the names of several British adventurers killed in the fight for Texan freedom. In the evening we were entertained by Mexican dancing and to dinner on the San Antonio river.

In contrast to this traditional Texan life, we saw the latest in military medicine, including a burns unit, at Brooke Army Hospital where the research was directed by Colonel Edward H. Vogel. We attended an excellent orthopaedic conference under the auspices of Colonel Harold S. McBurney.

The U.S.A.F. provided a thrill at the aerospace medical centre, where Major-General Otis Benson explained the problems of projecting man into space and introduced us to Sam, the monkey who had ascended fifty-five miles. At the Lackland Hospital Colonel Earl W. Brannon gave his experiences of arthroplasty of the fingers and Major James Leslie described an interesting axillary approach for shoulder repair, which avoided leaving the ugly scar associated with incisions in this region.

The civilian programme comprised visits to the Robert B. Green Memorial Hospital and the Santa Rosa Children's Hospital. At the former the high incidence of major trauma and at the latter of tuberculosis and deformities were related to the large Mexican population. Dr John Hinchey reported a remarkable personal series of 186 prosthetic replacements for femoral neck fractures in elderly patients.

After dinner with the Bexar County Medical Society the Fellows were asked to speak on the National Health Service and, in response to questions, held out for two hours.

Houston (*May* 19)—While house guests of Dr Ed Smith we were privileged to receive the Honorary Citizenship of Texas, presented by Judge McLelland on behalf of the Governor of the State.

In a crowded day we toured the newly built Texas Medical Centre, incorporating Baylor University Medical College. We saw a perfusion in progress for melanoma of the foot at the M. D. Anderson Hospital; Dr Smith showed cases of soft-tissue correction for flexor contracture of the hips and a group of child amputees. The Texas Institute for Rehabilitation and Research was remarkably equipped for continuous pulse, blood pressure, respiration and electrocardiograph recording and catered for those seriously crippled whatever the cause. The teaching of practical physiology was based on electronic techniques and all the information from the rehabilitation unit could be picked up by television in the physiology department in neighbouring Medical School buildings.

The afternoon programme included an account by Dr Paul Harrington of his method of instrumentation for the correction of scoliosis, and by Dr M. Donovan on atlanto-axial fusion.

We then took a voyage, with most of the orthopaedic surgeons in the city, down the Houston channel to view the oil refineries and chemical works. At San Jacinto we went ashore and toured the site of the famous battle before a dinner given by the Houston Orthopaedic Club of which we were made Honorary Fellows.

Galveston (*May* 20–21)—We were accommodated in a very modern hotel on the Gulf of Mexico as guests of Dr George Eggers and Dr Burke Evans. The programme was in the medical school of the University of Texas, which has interesting associations with Edinburgh. Dr Eggers traced his interest in the contact-compression principle in fracture treatment and this was amplified by a report on forearm fractures. He also discussed the surgery of cerebral palsy with particular reference to release operation at the knee. Dr Evans prepared an excellent demonstration of bone changes in burns and Dr Charles M. Pomerat showed a truly remarkable film of cell behaviour in tissue culture, a technique which seems likely to open new avenues of research.

Memphis (*May* 22–24)—Dr Andrew Crenshaw was among those who met us at the airport with an unusual conveyance, a London taxi, vintage 1935, which confederate members of the A.B.C. Club had provided as the answer to the " deep freeze " of the first American tour of Britain. Our hosts at the Campbell clinic had arranged a programme emphasising research in the University of Tennessee, including studies on radioactivity in bovine thyroids, tolerance to radioactivity in dogs, marrow transplants, sickle-cell anaemia, use of P^{32} in avascular necrosis and the rate of union after refracture in rats. At the clinic Dr Harold Boyd reported the results of bone-grafting for non-union and showed a film on pseudarthrosis of the tibia; Dr Marcus Stewart showed a film on anterior cervical fusion; Dr Lee Milford spoke on local skin cover in the hand and Dr R. A. Calandruccio on cartilage regeneration; Dr Fred Sage described his technique of intramedullary fixation of the radius and ulna.

We went on a ward round with Dr T. L. Waring at the Crippled Adults Hospital, and, at the Children's Hospital, Dr A. Ingram discussed the correction of relapsed club foot and the syndrome of congenital vertical talus and hallux varus; in each case Kirschner wire fixation was used to maintain correction.

Dr J. S. Speed was in the chair when we were entertained to dinner at an ante-bellum house and afterwards to singing by the negro waiters. On another evening each of the Fellows gave a short paper at the Clinic. On the final night we dined at the Country Club and were then entertained in characteristic manner at the house of Dr Hugh Smith, the music being provided by Beale Street before our host took over.

Chapel Hill and Durham (*May* 25–27)—Leaving Memphis at dawn, a series of flights brought us to the University of North Carolina in the afternoon, where we attended a scientific session under the chairmanship of our hosts, Dr Beverly Raney and Dr H. R. Brashear.

The following day, spent in Durham, started at the Cerebral Palsy Hospital with a clinical demonstration of the surgical treatment of cerebral palsy, Dr Lenox Baker discussing the lower limb and Dr Leonard Goldner the upper. Dr Baker stressed the importance of precise diagnosis of the elements making up contractures, while Dr Goldner gave convincing proof of the value of flexor tendon transfers with or without fusion of the wrist.

We visited a tobacco factory and toured the Campus of Duke University before attending a scientific session which included a discussion of the surgery of relapsed club foot by Dr Goldner.

Another day was spent in Chapel Hill touring the University and taking part in a scientific programme, during which we received the glad news that a third Doig son had safely arrived, who was later accorded, *in absentia*, the unique privilege of Honorary Junior Membership of the American Orthopaedic Association.

Hot Springs (*May* 28–*June* 2)—The climax of our tour, the meeting of the American Orthopaedic Association, was held in the beautiful surroundings of the Allegheny Mountains. A report of this will be found elsewhere. Suffice it to say that the kind greetings of the President, Dr John Royal Moore, and the warm welcome accorded to us by those we now thought of as old friends, touched us deeply, and the time passed all too quickly. We were specially glad to meet Professor R. I. Harris, Dr Neil Garber and members of the exchange committee of which Dr William Schnute is chairman.

Washington (*June* 3–4)—Two wonderful days were spent sightseeing as the house guests of Dr W. Tobin, Dr Alan McKelvie and Dr John Adams. We particularly enjoyed visiting Mount Vernon and walking through Georgetown.

New York (*June* 5–8)—During the last few days we enjoyed the hospitality of Dr F. R. Thompson. No official programme had been arranged, but we each made contact with various branches of orthopaedics according to our particular interests, and some members visited Toronto.

What do we learn from such a tour? A great deal of practical orthopaedics, an understanding of American training and practice, an awareness of our own shortcomings in matters of pathology and research, a remarkable similarity of clinical problems, useful details of building, organisation and equipment, amongst ourselves some personal self-discipline. . . . Then there are the places, and the cameos they leave in the mind; evening traffic by Lake Michigan, dawn sunlight on the Rockies, the magic carpet of San Francisco seen at night from the air, the blinding heat and parched landscape in a momentary stop at El Paso, ships on the Mississippi, the Englishness of Virginia, the Americanness of the receding skyline of lower Manhattan. . . . Above all it is the people, the wonderful hospitality, kindness and thoughtfulness of our hosts, who saw us leave America fitter than when we arrived. Again details crowd the mind; the rush for the plane at Chicago, photographs at the Mayo clinic, a night-cap on the "C.P.R.," trophies of the chase on study walls, children, white and coloured, Eskimo and Indian, Mexican and Chinese, laundry sessions at midnight, a well known figure vacillating between piano and organ, two more paying a nocturnal visit at the Homestead . . . for all these things and many more we are grateful to the British and Australian Orthopaedic Associations who sent us; and to the members of the American and Canadian Associations, their wives, and all their local clubs who provided for us and arranged the tour.

J. H. S. SCOTT,
J. W. DICKSON,
W. G. DOIG,
A. H. C. RATLIFF,
R. H. C. ROBINS.

DAME GEORGIANA BULLER MEMORIAL LECTURE

The third Dame Georgiana Buller Memorial Lecture was given by Sir Harry Platt, *Bart.*, at the Annual General Meeting of the British Council for Rehabilitation on board H.Q.S. *Wellington*, Victoria Embankment, London, by kind permission of the Master and Members of the Honourable Company of Master Mariners, on May 12, 1960. His subject was "Pioneers of Rehabilitation."

After paying tribute to the life and work of Dame Georgiana Buller in the cause of the physically disabled, Sir Harry described the evolution of rehabilitation throughout the ages from the time of Hippocrates to the present day. Rehabilitation was not, as had been often said in recent times, a "child of the second world war." Amongst the pioneers mentioned by name, with a brief reference to their achievements, were Nicholas Andry, Sir Robert Jones, Dame Agnes Hunt, Sir Henry Gauvain, Konrad Biesalski, J. E. Goldthwait, and G. R. Girdlestone. There was, however, a great company of unnamed pioneers whose contributions would not be forgotten. In this category, apart from individuals, were the voluntary organisations; as also were central and local governments by means of social legislation.

The pattern in Great Britain was becoming increasingly complex because of the existence of many organisations devoted to the welfare of the physically handicapped. There was much overlapping of effort and competition for charitable funds. For these reasons Sir Harry felt a national council was needed to act as an adviser and coordinator.

SCIENTIFIC SOCIETIES AND REGIONAL ORTHOPAEDIC CLUBS

THE BONE AND TOOTH SOCIETY

A meeting of the society was held at the Nuffield Department of Orthopaedic Surgery, Oxford, on March 29, 1960.

Chemical constituents of bone matrix—*Dr P. W. Kent* (Oxford) reviewed the mucopolysaccharides known to occur in the organic matrix of bone, and described the structure of chondroitin sulphates A and C. These two highly acidic polymers were unbranched chains comprised of alternating residues of N-acetyl-D-galactosamine and D-glucuronic acid. The two substances differed in the position of the sulphate ester group which, in type A, was located mainly at C_4 of each aminosugar unit and, in type C, at C_6. Available analytical methods did not preclude the presence of further forms of chondroitin sulphates having the sulphate groups distributed in other positions. The biochemical origin of galactosamine and glucuronic acid was traced from D-glucose and a mode of polymerisation involving their uridine diphosphate nucleotides was considered. Recent results suggested that the carbohydrate skeleton was first formed (either partly or in full) and that this was enzymically sulphated through "active sulphate" formed from adenosine triphosphate and SO_4^{2-}. The sulphation step appeared to be dependent on the nutritional state of the intact organism and in particular on the ascorbic acid level.

Other mucosubstances found in bone included complex polymers containing a variety of neutral sugars (galactose, mannose, glucose, fucose). One such, extractable from bone during decalcification with ethylenediamine tetra-acetic acid, had a positive periodic acid-Schiff reaction and might be one of the agents responsible for this histological feature of this tissue.

Changes in the organic matrix of enamel during growth—*Dr M. V. Stack* (Bristol) discussed the quantitative changes in the amounts of matrix present in developing enamel fractions which were examined after separation by flotation from human dentitions. Organic content was related to arbitrary specific gravity intervals defining the enamel fractions. Moisture and organic content were proportional in the enamel of various developing crowns in which the cervical margin of the enamel had not been completed. The rate of "deproteinisation" of enamel during mineralisation was deduced from a curve showing the amounts of enamel protein in known total weights of enamel in dentitions at various ages. All the enamel from the deciduous dentition might finally contain as little as 10 milligrams of protein, but this was only one-tenth of the amount present one month after birth. Fractionation of the organic components demonstrated that acid-soluble protein accounted for about one-third of the total in enamel separated at specific gravities between 2·2 and 2·4, and between 2·4 and 2·6, as had already been shown in the case of mature enamel. The proportion of diffusible peptide material increased during the stages associated with the tenfold decrease in total protein content, while that of insoluble protein decreased.

The fine structure of bone, cartilage and tooth matrix—*Dr K. Little* (Oxford) showed electron microscope photographs of fibrous tissue, cartilage, bone and dental enamel. She said that in tissues such as the intervertebral disc, with collagen fibrils associated with cell walls, these walls were thick and prominent. In developing cartilage, which had collagen precipitated away from the cell walls, they were very thin. Sections of tendon containing the cellulose-like fibres described by Cruise and Keech (*Journal of the Royal Microscopical Society*, 1958, **78,** 95) were badly torn, presumably because of the relative hardness of these fibres. A few areas of abnormal cartilage had been found, with early calcification showing as rosettes of dense crystallites. Normal apatite deposits had a less dense appearance.

Sections of decalcified bone were next shown. When decalcification was preceded by formalin fixation, the state of preservation was good. Normal bone, decalcified without fixation, showed a reasonable preservation of the matrix, with its architecture remaining intact. Normal bone from old people also had a well preserved matrix. A bone decalcified after 1,800 years showed good preservation of the collagen fibrils of large diameter, and also of part of the ground substance. By contrast, decalcified osteoporotic bone showed a formless and degraded matrix.

In developing dental enamel the matrix of the prism walls was seen to be formed between ameloblasts and the matrix of the core at their ends. The newly formed matrix showed less structure than that in the immediately pre-calcified state. Normal enamel which had been treated by the Brain technique (with a solution containing formic acid) showed that the matrix of the prism walls—and consequently the apatite contained therein—was unaffected, while the matrix of the prism cores was removed. Sections of the highly fluorosed teeth described by Darling and Brooks (*Journal of Dental Research*, 1959, **38**, 1,226), similarly treated, showed that there was either no solution of the bulk of the matrix, with retention of all the apatite, or, in the middle portion of the enamel, that the prism walls were dissolved. It was thus confirmed that the action of the fluorine had been to influence the composition and properties of the matrix.

Histological abnormalities in the matrix of bone and cartilage—*Dr J. W. Landells* (London) said that histological observations on the formation of bone matrix illustrated the importance of the polysaccharide component, though this was shown by bulk chemical analysis in small quantity only. In osteogenesis imperfecta the formation of early polysaccharide-rich bone was possible, but not the later formation of collagen-rich bone, though collagen elsewhere in the body was unaffected. The rare condition of fibrogenesis imperfecta was largely a defect in the synthesis of properly orientated collagen; but this did not calcify, and the distribution of polysaccharide was again abnormal. Paget's disease showed the detail of the bone only slightly deranged, but the adhesion of structural elements was weak, and the balance between formation and destruction much too labile; there was a demonstrable tendency to the resorption of calcified material; this must precede the formation of abnormal bone and was therefore likely to be the basic abnormality. Osteopetrosis was the reverse; an inability to destroy calcified cartilage initially handicapped bone formation, and similar difficulty attended the action of osteoclasts in removal of bone; there was an associated defect in the manufacture of the adhesion material. In all four examples of abnormal matrix the polysaccharide was intimately concerned.

Disintegration of joint cartilage—*Professor J. Trueta* (Oxford) said that the clinical and experimental work on which his department had been engaged for the last ten years suggested that osteoarthritis was a degenerative process brought about by any of the several mechanisms which affected the vitality of the chondrocytes of the articular cartilage. Although the chondrocytes might suffer or die from changes in the composition or amount of joint fluid it was far commoner for this to occur from the wrong, and thus damaging, way in which joint cartilage was subjected to intermittent compression. Sometimes this faulty joint function depended on anatomical alterations of the shape of the joint; at other times it was wrong joint function which caused part of the joint cartilage to be excessively compressed while the rest was insufficiently under the compressing forces. The whole of the changes that followed the damage to the cartilage cells might be explained as a repeated reparative effort manifested particularly in the vessels underlying the cartilage in the bone epiphyses.

Experimental evidence of these facts had been gathered some years ago by Dr A. Trias, and his paper was now in the course of publication. By compressing the joint with the use of a small apparatus based on that used by Charnley in his technique of compression arthrodesis, all the pathological changes present in osteoarthritis of man, from osteophytes to bone cysts, had been produced. It also became apparent that the degenerative process was reversible, for new chondrocytes appeared covering the bone surfaces of the joint in these experiments when the compressing forces were removed.

The organic matrix of the dental enamel in some diseases—*Professor A. I. Darling* (Bristol) said that the histology of dental enamel during development was described in three stages: 1) a relatively structureless sheet; 2) differentiation of prism sheaths or cortices; 3) an apparent loss of the matrix from the core of the prism. The reduction of the matrix in the early stages might be due to some chemical or physical difference, but could also be due to a more easy access of fixatives. In fluorotic enamel and the enamel of amelogenesis imperfecta, the matrix of the hypocalcified areas corresponded in appearance to the two earlier stages. This was open to the same interpretation as that given for developing enamel. In dental caries it was shown that the earliest demonstrable attack on the enamel, that is the translucent zone, showed the presence of spaces without recognisable decalcification. It was also shown that the enamel contained two types of structure, one readily dissolved in the carious process and the other relatively resistant to attack. From these two points it was argued that the earliest attack was probably caused by a solution of the soluble organic matrix of the enamel, and that this

soluble matrix was probably distributed in the structures readily attacked by caries, while the more resistant structures contained a matrix of insoluble organic material.

The chemistry of intervertebral disc prolapse—*Dr N. G. C. Hendry* and *Dr P. E. G. Mitchell* (Aberdeen) said that fifty normal and fifty prolapsed discs had been analysed for their polysaccharide content (as hexosamine and reducing sugar), for collagen (as hydroxyproline), and for total nitrogen. The mean figures in all estimations were significantly different, with, in the prolapsed discs, a fall of polysaccharide (4·23 milligrams of hexosamine per 100 milligrams dry weight against a normal of 6·99 milligrams; 11·15 milligrams of reducing sugar against a normal of 6·45 milligrams), a rise in collagen (43·75 milligrams, the normal being 24·88 milligrams), and a rise in total protein (11·62 milligrams, the normal being 8·54 milligrams). In the normal disc there was a slow rise and then a fall in hexosamine levels, the maximum being in middle life, and a slight increase in collagen throughout life. In the prolapsing disc, changes—which probably began about eighteen months before the first onset of symptoms—consisted in a sharp fall in polysaccharide and a similar rise in collagen. These changes were unrelated to age, but had a direct relationship to the duration of the disability. In both the normal and the "prolapsing" sequences nitrogen levels remained constant, in spite of the changes which were occurring in collagen and, presumably, in polysaccharide-bound protein. The significance of this finding was not known.

Changes in the bone matrix—*Dr M. E. Kelly* (Oxford) and *Dr A. Courts* (London) had made an investigation to determine the changes in collagen in ageing and osteoporosis. The bone matrix from normal human femurs from birth to eighty-seven years, from femurs showing macroscopic and histological signs of osteoporosis, and from femurs showing marked ageing, was examined. After defatting and demineralisation, the bone was treated with alkaline solution, after which one component of collagen from normal bone became soluble in citric acid, while the other remained insoluble. Various collagen fractions were studied by x-ray diffraction. In the soluble collagen of normal bone the rings were closer together than were those in the 4–5 Å region of the insoluble fraction. These differences in position and intensity seemed consistent with a real chemical difference. In elderly bones the processing resulted in the hydrolysis of fatty acids, which gave intense rings superimposed on the collagen picture. In bone matrix from osteoporotic bones the insoluble portion gave a third variant of the collagen diffraction picture. By chemical separation and x-ray diffraction and electron microscopy examination the matrix of osteoporotic bone could apparently be distinguished from the matrix of normal bone of any age.

A further meeting of the Bone and Tooth Society was held at the Royal National Orthopaedic Hospital, London, on July 6, 1960.

Avian osteopetrosis—*Dr J. R. Holmes* (Bristol) said that this was a sporadic virus disease of the fowl characterised initially by a progressive periosteal proliferation resulting in thickening of the diaphyses of the long bones, and that later both the endosteum and the compacta were involved; finally the whole skeleton might be affected by the lesions which were usually bilateral and symmetrical. The disease became apparent when involvement of the metatarsals caused visible thickening of the legs at the age of two to three months.

Osteopetrosis could be induced by the intraperitoneal injection of day-old chicks with filtered plasma from birds with active bone lesions, and observations on the radiological changes in experimentally induced osteopetrosis in 108 birds killed between five and ninety-seven weeks of age showed that the changes in the long bones could be classified into four main types: *Type A*—Periosteal proliferation followed by lesions in the endosteum and the compacta—49·1 per cent. *Type B*—Very advanced proliferative changes with widening of the bone, a very dense compacta and virtual obliteration of the marrow cavity—17·6 per cent. *Type C*—Sclerotic changes in the compacta and radio-opaque areas in the endosteum with minimal periosteal reaction—13·9 per cent. *Type D*—Usually a sequel to type A or B and characterised by the development of osteolytic foci in the bones —19·4 per cent. This type was seen only in birds over twenty weeks of age.

The pectoral girdle was affected in 91 per cent of birds and the lesions resembled those in the long bones; of these bones the clavicle was invariably affected and the scapula the least. In the pelvis lesions occurred less frequently and were mainly proliferative, involving particularly the lateral walls of the pelvis. The vertebrae were occasionally involved, and the digits and the skull were affected in some birds over twenty-three weeks of age, characterised mainly by periosteal proliferation. The joints were normal. Measurements of affected long bones showed shortening and an increase in width when compared with normal controls.

The influence of citrate on the composition and form of calcium phosphate precipitates—*Dr T. C. A. McGann* (Cork) said that the so-called colloidal phosphate of milk had been shown to possess a

high citrate content intimately associated with the phosphatic constituent and comprising some 12 per cent by weight of the total complex. The calcium-phosphate-citrate precipitates formed on neutralising acidified milk sera were basically similar in composition. Both could be represented approximately by the empirical formula of the hypothetical citrate apatite, $3Ca_3(PO_4)_2CaHCitr$. The phosphate-citrate precipitates formed in milk sera were found, moreover, to exhibit typical apatite x-ray diffraction patterns.

The presence of citrate in solution from which calcium phosphate was being precipitated tended to induce the formation of a more basic phosphate precipitate than would otherwise occur at the same pH and calcium ion concentration; the relatively high binding power of citrate to calcium might be involved in this. The binding of citrate in the resulting apatite-like crystals was presumably similar to that of carbonate in the microcrystals of bone.

The possible physiological implications of these findings were briefly discussed in the light of the increasing recognition given to the role of citrate in the process of calcification.

Variations in the structure of normal bone—*Dr J. Jowsey* (London) had surveyed the structural changes related to age in forty-seven individuals from one and a half to ninety-three years of age using normal bone from the centre of the shaft of the femur. Microradiographs of thin calcified cross-sections were measured for information on the quantity of bone turnover in an area that comprised one-third of the entire cross-section.

Bone turnover took place by bone formation and resorption and, since this occurred on the surface of bone, the amount could be measured. The porosity of the bone had been estimated by counting the number of osteones that were incompletely closed, the number of Haversian canals filled with calcified material and the number of osteocyte lacunae filled with bone mineral.

The results showed the variations in structure of cortical bone at different ages. Children showed a high turnover, young adults showed remarkably little activity and the bone had a compact appearance with little variation in mineral density. Later in life there was an increase in the amount of resorption while bone formation remained at about the same level. In old age there was a further increase in bone porosity largely because of incomplete closure of osteones; and there was also an increase in the number of plugged canals and filled lacunae.

Chemical composition and histological structure of bone—*Dr C. Fabry* (Liége) said that compact adult bone was structurally heterogeneous and the mean dimensions of the histological units were about 10,000 times greater than the mineral crystallites; and that she had attempted to correlate chemical composition and histological structure. First the refractive index of single dissected osteones was measured; but this was time consuming, and too many dissections were necessary to obtain accurate chemical information; this led to a search for a method of fractionation based on the variable physical properties of bone such as " grindability " and specific gravity. Diaphysial adult bone was ground in different conditions, and the weight distribution obtained by sieving was correlated to the particle size. A normal distribution was always obtained, indicating that bone tissue broke without any preferential direction. Calcium, phosphorus and carbon dioxide percentages were the same in each fraction. However, different fractions were obtained by flotation of bone powder in liquids of variable densities, which corresponded to young or old tissue. The increase of density corresponded to the rise of the refractive index and also of the ratio of inorganic to organic material. Unexpected chemical variations also appeared: the percentage of calcium increased proportionally more than that of phosphorus, in such a way that the calcium/phosphorus ratio increased with the density; the CO_2/P (or CO_2/Ca) ratio also increased, but the percentage of magnesium seemed to fall.

A microscopic method of determining rates of bone growth—*Dr W. H. Harris* (London) had studied intravital staining of growing bone surfaces in man, dogs and rats, using a variety of agents including alizarin red S, chlorazol fast pink, chlorazol sky blue, calcium fast purple, naphthamine brilliant blue RR and certain tetracyclines (tetracycline, chlortetracycline, oxytetracycline and demethylchlortetracycline). The tetracyclines were by far the most satisfactory because they were selectively incorporated into bone tissue on surfaces where there was active bone formation, and because they could be identified readily in calcified sections by fluorescence to ultraviolet light; also, unlike the other agents such as alizarin red S, radioactive isotopes and lead salts, they were effective in non-toxic doses.

In the dog a single intravenous injection of 20 milligrams per kilogram of oxytetracycline was a satisfactory marker of growing bone surfaces. Two oral doses of 1 gramme of any of the tetracyclines six hours apart also gave a similar marking in human bone. The rate of accretional bone growth could be determined by giving two or more marker doses on separate occasions and measuring the distance between the lines of tetracycline uptake. In one young dog accretional growth rates of approximately seven to fifteen μ a week were found in the developing osteones of the distal part of the cortex of the fibula.

The use of tritiated thymidine in the study of bone—*Mr N. F. Kember* (London) had found that when thymidine was injected into animals it was only incorporated into cells that were synthesising desoxyribonucleic acid. Tritium-labelled thymidine could, therefore, be used to label and detect—by tissue section autoradiography—cells that were in synthesis at the time of injection. The tritium labelling was permanent, and labelled cells had been followed by autoradiographs for a year after injection and through two or three divisions. A study of endochondral ossification in rat tibiae was made in this way. Immediately after injection labelled cells were found distributed at random across the columns of the upper half of the epiphysial cartilage plate. Two days after injection labelled nuclei were found in the region of hypertrophied cells which gave a measurement of bone growth. The generation times of the dividing cartilage cells could be estimated and a region of reserve cells defined at the top of the cartilage plate. In the metaphysis, shortly after the injection, only progenitor cells were labelled, with an occasional osteoblast. After one to three days increasing numbers of labelled osteoblasts were seen, as were labelled nuclei in osteoclasts. This indicated that osteoblasts and osteoclasts were both formed by differentiation from a progenitorial mesenchymal cell.

Dr J. MacGregor (Glasgow) and *Dr B. E. C. Nordin* (Glasgow) also read papers.

EAST ANGLIAN ORTHOPAEDIC CLUB

A meeting of the East Anglian Orthopaedic Club was held at the Norfolk and Norwich Hospital on Saturday, March 26, 1960.

Artificial joints—*Mr G. K. McKee* discussed his indications for arthroplasty and made the point that, if pain could be relieved, a movable hip must be better than a fixed one. He discussed his artificial hip joint; he was now using a Thompson prosthesis made in Vitallium working in an artificial acetabulum. A number of patients treated with different prostheses were demonstrated, as was an artificial knee joint and patients in whom it had been used successfully.

Leri's disease—*Mr G. D. Farrington* (introduced by Mr R. Howard) showed a man aged forty who had pain, swelling and limitation of movements of his right knee. During the war he had had polyarthritis involving many joints but only the knee continued to give trouble. There was a mass displacing the patella. Radiographs showed " candle-grease " deposits down the femur and on the tibia. This mass was removed and was found to consist of bone and cartilage.

Congenital absence of radius—*Mr J. G. Taylor* showed a small child with congenital absence of radius with deformity of the wrist so that there was inadequate dorsiflexion and a poorly controlled thumb. The child could, however, use the hand. In the discussion on treatment it was agreed that improving the appearance might damage the function.

Congenital coxa vara—*Mr J. G. Taylor* showed a child with congenital coxa vara which presented at the age of five and which looked like a congenital dislocation of hip. Radiographs showed the typical deformity and also a well marked additional centre of ossification below the femoral head. Treatment by osteotomy and wide abduction had left the child with some shortening of the femur.

Thompson's prosthesis—*Mr W. Kellock* considered the value of a Thompson Vitallium prosthesis in treatment of fractures of the femoral neck both as a salvage operation and also in recent high fractures or those with gross displacement. The results in a series of sixty patients whose average age was seventy-four were satisfactory. Various approaches had been used—and there had been dislocations with each—but good functional results were obtained and the patients could be out of hospital in six weeks.

Dislocation of the hip—*Mr W. Raffler* discussed the problem of dislocation of the hip discovered late after multiple injuries and mentioned patients in whom the dislocation was discovered four to five months after the injury. He advocated radiographic examination of both hip joints when there was a fractured femur to prevent this risk.

Haemangioma—*Mr R. Howard* demonstrated patients with haemangiomata of the vertebral body and of the knee joint. The former occurred in a girl of eleven who first complained of pain after falling off a swing and hurting her back. There was a surprising amount of muscle spasm and she was treated in a plaster bed for six months with complete relief. Three months later pain recurred and she developed a cauda equina lesion and radiographs showed some vertebral collapse. Exploration was cautiously carried out with a needle and this was followed by a " monumental haemorrhage." A bone graft was inserted to fix the body above and below the lesion and she was immobilised in a plaster bed. She still had paraplegia and was to be given radiotherapy. The second haemangioma was in a girl of eleven who had repeated effusions in a knee that, on aspiration, showed bloodstained fluid. The radiograph remained consistently normal. At exploration a haemangioma was found and removed.

Thoraco-scapular arthrodesis—*Mr R. Howard* showed a film of thoraco-scapular arthrodesis for extensive paralysis of the shoulder girdle.

SHEFFIELD REGIONAL ORTHOPAEDIC CLUB

A meeting of the Sheffield Regional Orthopaedic Club was held at Grimsby General Hospital on March 26, 1960.

Osteochondritis dissecans of the knee—*Mr S. M. Thompson* and *Mr N. E. James* showed three patients. In one the lesion had healed after simple drilling. In the second, spontaneous healing of the lesion had taken place. The third patient had large lesions in both knees but had declined operation. In spite of this he had very little disability.

Traumatic dislocation of the hip—*Mr S. M. Thompson* showed four patients with dislocation of the hip which could not be reduced by closed manipulation. The factors which obstructed reduction were a detached fragment of the femoral head lying in the acetabulum, inturning of the joint capsule and osteophytes in an osteoarthritic hip, interposition of muscle, and detachment of the posterior rim of the acetabulum with a sciatic nerve lesion.

Glomus tumour of biceps femoris—*Mr N. E. James* discussed a twenty-six-year-old woman with a ten-year history of pain at the back of the right knee. Nothing had been found at previous exploration some years ago, but at a recent operation an oval mass was removed from the lower part of the biceps muscle. The consensus of opinion on the histological diagnosis was in favour of a glomus tumour.

Bacterium mycoides arthritis of the left knee—*Mr N. E. James* showed a patient with this infection following a prick by a thorn. The diagnosis had been made by culture of the joint fluid and was confirmed by blood agglutination tests. The organism was sensitive to penicillin and sulphatriad and the infection had responded to conservative treatment.

Familial osteopetrosis—*Mr S. M. Thompson* showed five young men—from a family of ten—of whom four suffered from osteopetrosis. The fragility of the bone, with rapid union after fracture, and the liability to chronic osteomyelitis and sequestrum formation were shown. The fifth member of the family suffered from spondylolisthesis but not from osteopetrosis.

Dislocation of the cervical spine—*Mr Thompson* also showed a patient in whom skull traction had failed to reduce a dislocation of the cervical spine and manipulation under an anaesthetic was required. This was successful and partial recovery had taken place so that the patient could now walk with crutches and calipers.

Fracture of the medial epicondyle of the elbow—*Mr N. E. James* showed two patients who had sustained fracture-dislocation of the elbow with the medial epicondyle caught in the joint. In each the medial epicondyle had been reduced out of the elbow joint by closed manipulation and operation had not been necessary.

IRISH ORTHOPAEDIC CLUB

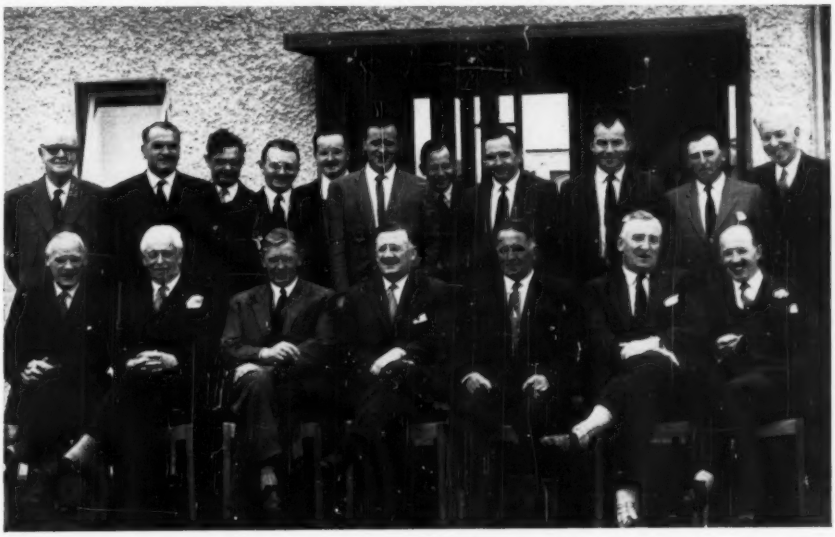
The spring meeting of the Irish Orthopaedic Club was held at the Orthopaedic Hospital, Kilkenny, on May 28, 1960.

Rural orthopaedic problems—*Mr R. F. O'Driscoll*, the Club's host on this occasion, spoke of the orthopaedic problems peculiar to a rural area with its long distances, inadequate after-care and great shortage of junior medical staff. These problems affected particularly the treatment of congenital club foot, where great difficulty was experienced in maintaining the correction achieved in the early stages.

Treatment of relapsed club foot—*Mr F. Dwyer*, guest speaker from Liverpool, read a stimulating paper on the treatment of difficult or relapsed club feet, for which he had devised an operation in which a medial osteotomy of the calcaneum, held open by a tibial bone graft, was used to correct the inversion and increase the height of the heel. He claimed that, by this procedure, the balance of the foot was improved and weight bearing became a correcting instead of a deforming force. Mr Dwyer's paper was illustrated by slides and a most convincing film.

Intertrochanteric osteotomy—*Mr B. F. Regan* (Kilkenny), after reviewing a series of patients with osteoarthritis of the hip and femoral neck fractures treated by intertrochanteric osteotomy and internal fixation, gave as his conclusion that fixation by the McKee nail-plate gave results at least as good as, and in some respects better than, plaster fixation.

Congenital dislocation of the hip—*Mr J. Kelly* (Limerick) reviewed the literature on the spontaneous cure of congenital dislocation of the hip, and described one case of his own.



Irish Orthopaedic Club—Spring Meeting, May 1960—Kilkenny. The senior members in the front row are, from left to right, J. C. Cherry, Arthur Chance, F. Dwyer, N. S. Martin, R. F. O'Driscoll, St J. O'Connell, P. A. M. Fitzgerald.

Clinical demonstration—In the afternoon the members toured the new hospital at Kilkenny and were much impressed by the layout and modern facilities in the various departments. At a clinical demonstration many very interesting patients were shown and discussed.

A short visit was made to the Orthopaedic Department of the Waterford Hospital, and the day ended with an informal dinner at Tramore.

SOUTH AFRICA

SOUTH AFRICAN ORTHOPAEDIC ASSOCIATION

The annual congress of the South African Orthopaedic Association was held in East London from September 28 to October 2, 1959. The meeting was presided over by Mr J. J. Commerell, and distinguished guests included Miss Maud Forrester-Brown (Edinburgh) and Dr Rex L. Diveley (Kansas City).

The following were elected office-bearers of the Association for the ensuing two years: *President*—Mr R. C. J. Hill (Durban), *Honorary Secretary*—Mr C. J. Kaplan (Durban), *Treasurer*—Dr Mungo Thompson (Pietermaritzburg), *Members of the Executive Committee*—Mr A. J. Helfet (Cape Town) (*Past President*), Mr J. G. Bickerton (Durban), Mr J. J. Commerell (Cape Town), Mr F. J. Hedden (Durban), Mr N. H. Louw (Bloemfontein), Mr L. Mirkin (Port Elizabeth), Mr C. T. Moller (Johannesburg) and Mr W. T. Ross (Johannesburg). It was agreed to appoint Mr George Dommisse the Archivist to the Association.

The diagnosis and treatment of the painful back—*Dr Rex L. Diveley* (Kansas City) said that in an analysis of some 1,000 individuals in his country he had found that over 45 per cent of persons over the age of forty have, or have had, some symptoms of backache, and approximately 42 per cent had some congenital anomalies and weakness of the lumbo-sacral spinal structure. He pointed out that the transition from the quadrupedal gait had left the spinal segments in a position in which they were architecturally weak, and that the oblique position of the sacrum increased the mechanical strain of the lower lumbar segments. Dr Diveley discussed in detail the numerous points to be elicited in the history of low back pain, and described the clinical examination of the spine and ancillary investigations. In patients with true low back pain with symptoms of root pressure he advised simple laminectomy and nerve root decompression, fusion being reserved as a secondary operation when relief was not obtained at first. The combined operation of fusion and root decompression was reserved

for patients with an unstable spine, especially with an unstable lumbo-sacral joint with congenital abnormalities. Finally, Dr Diveley said that he considered that a prolapse of the intervertebral disc was generally the result of a strain or sprain of unstable spinal segments, and emphasised the importance of examination of the low back before employment in industry.

Operative treatment of prolapsed lumbar discs—*Mr G. T. du Toit* and *Mr D. Roux* (Johannesburg) discussed their results in fifty patients treated by operation over a period of five years. The predisposing factors to disc protrusion were vertebral epiphysitis, a poorly developed posterior longitudinal ligament, spondylolisthesis, and sacralisation of the fifth lumbar vertebra; but, in 50 per cent of the patients, an acute traumatic prolapse followed bending and lifting strains. The importance of local tenderness over the interlaminar space on either side of the midline was stressed as being a most valuable sign in deducing the level of the prolapsed disc, and in distinguishing between prolapse at the lumbar 4/5 and lumbo-sacral levels. It was felt that spinal manipulation was particularly dangerous, as being liable to cause acute compression of the cauda equina from sudden displacement of a large mass of disc material; and that care should be taken to exclude the presence of either a tumour of the cauda equina, which may closely simulate a disc lesion, or obliterative vascular disease of the aorta or its bifurcation. Conservative treatment was given in all patients—apart from those with acute cauda equina compression—for six weeks, and positioning a patient in a low Fowler position was preferred to the more commonly practised leg traction with weights. The indications for operation were discussed and, in passing, it was noticed that several young patients (ten to twenty years old) failed to respond to prolonged conservative treatment and required surgical intervention. At operation the Mohammedan praying position was used, and the disc explored through a bilateral interlaminar approach. Spinal fusion was performed at the same time in five of the fifty patients, in whom there was instability at the level of the disc lesion, spondylolisthesis, or a collapsing type of back in a willowy young lady. The results showed that, of the fifty patients, twenty-five had complete relief of all signs and symptoms; sixteen had only a moderate degree of backache after strenuous exercise, but were otherwise good; and five had only relief of sciatica, but moderate backache continued. There was only one bad result in which immediate fusion had been performed, and the graft was later found to be pressing on the spinal cord.

The cervical disc syndrome—*Mr R. C. J. Hill* (Durban) said that it was now recognised that brachial neuritis, especially in the aged, was due not only to cervical disc degeneration, but also to degeneration of the neuro-central joints of Luschka, with secondary narrowing of the intervertebral foramina and consequent nerve root irritation. One hundred and fifty patients with pain in the neck or pain referred to the arms had been reviewed. The degenerative changes of the neuro-central joints were greatest between the fifth and sixth cervical vertebrae, and next in frequency came the joints between the fourth and fifth, and sixth and seventh, cervical vertebrae respectively, usually with degeneration of the corresponding disc. Both sexes were equally affected and the changes were greatest in the fifth and sixth decades, suggesting that this was due to a normal ageing process rather than trauma, although the latter might be a precipitating factor. Pain in the neck or neck and arm was the commonest symptom, radiating occasionally to the head, face, eye and ear, accompanied by stiffness of the neck, and frequently by weakness, numbness and pain in the hands. Five per cent of patients had symptoms referred to the lower extremities. The over-anxious middle-aged woman or the overwrought business executive was often found with these symptoms, and other patients had poor posture or performed unusual activities at the week-end. In these patients repeated small injuries induced the condition, as opposed to others with an acute injury—that is, hyperextension or whip-lash injuries of the neck. In 20 per cent of patients, nervous tension was a major symptom; and in this group coronary disease must be excluded. In long-standing cases with severe neck pain a local hypertrophy of the sterno-mastoid muscles was noticed. Treatment was rest on a firm bed with a low pillow, sedation, and intermittent cervical traction, followed by the wearing of a cervical collar. In established cases cervical manipulation was useful.

Several speakers were interested in the medico-legal implications of the so-called whip-lash injury: they felt that this term tended to create in the layman's mind a somewhat exaggerated picture of the mechanism. It was this mental picture, rather than the symptoms exhibited, which encouraged somewhat fanciful claims for damages. *Dr R. L. Diveley* pointed out that with the volume of traffic on the American roads, often bumper to bumper, the whip-lash could provide a means of potential financial gain for many sufferers and this prospect was exercising the minds of insurance companies.

Fracture of scaphoid bone—*Dr Rex L. Diveley* (Kansas City) pointed out the importance of the blood supply of the scaphoid bone after a fracture, and said that avascular necrosis occurred in 20 per cent of fractures of the proximal pole. Early diagnosis was essential, and after a severe sprain the wrist should be immobilised in plaster for ten days, and then further radiographs taken to exclude

a fracture. Fractures of the distal pole were immobilised in plaster for four to six weeks and all united satisfactorily, and fractures of the waist were immobilised for ten to twelve weeks. If avascular necrosis of the proximal pole occurred the affected portion was excised early to prevent arthritic changes. With established non-union it was better for the patient to accept the pain and discomfort and to find a job suitable to the wrist. When operation was performed excision of the proximal half of the scaphoid bone gave the best results, whereas excision of the radial styloid or bone grafting had been disappointing. Arthrodesis of the wrist was performed in selected patients with advanced osteoarthritis and had given favourable results. Dr Diveley quoted a series of 433 fractures treated in the American Army during the war, 320 of which had been followed up with 95 per cent of excellent results obtained by routine immobilisation in plaster. Dr Diveley had reviewed 124 consecutive fractures in his own clinic with very similar results.

Mr R. C. J. Hill (Durban) wondered whether, during the later years of the war, this fracture had not been given too much prominence, and thus overtreated. It was surprising to find how often an old ununited fracture was found on routine radiography, with little incapacity to the individual. *Mr A. J. Helfet* (Cape Town) recalled the operation of local fusion of scaphoid bone to capitate, which gave stability to the radial side of the wrist without impairing most of the function of the wrist. He had found this operation of value even in working men. Standard procedures for fusion of the scaphoid bone were discussed, but the past enthusiasm for this seemed to be on the wane.

Recent advances in cerebral palsy—*Dr B. Epstein* (Pretoria) said that since the first school in South Africa devoted entirely to cerebral palsy had been opened in Johannesburg in 1949 there had been an amazing growth of interest, and many other centres had now been opened throughout the Union. Research was being done throughout the world, and Professor B. E. F. Nel, of the University of Pretoria, had recently published a psychological and educational study of the cerebral palsied child. It was now possible to diagnose cerebral palsy at an earlier age, and research into its causes suggested that pre-natal factors were the most common. The treatment developed by Dr and Mrs K. Bobath of London, England, in which the body was dealt with as a whole, and attempts to overcome defective postural patterns were followed by physiotherapy, was being used extensively. The importance of chemo-pallidectomy and the more commonly used orthopaedic techniques in the correction of existing deformities were also mentioned. *Mr A. J. Helfet* (Cape Town) congratulated Dr Epstein on his presentation of a difficult subject that most orthopaedic surgeons tended to neglect. *Mr J. Craig* (Johannesburg) said that surgery had a definite value in the correction of established deformity.

Diametric fractures of the pelvis—*Mr G. F. Dommisse* (Pretoria) read his paper on this subject which was published in the last British issue (*Journal of Bone and Joint Surgery*, **42–B,** 432).

The problems of tuberculosis in the Bantu as seen by an orthopaedic surgeon—*Mr R. Percy-Lancaster* (East London) said that in the Ciskei and Transkei there were 2,000,000 Bantu. Their mode of life had remained relatively unchanged for many generations and ignorance, superstition and witchcraft were still all too common. The local diet consisted of some form of mealies, and malnutrition was found to be by far the commonest disease. Poor housing and hygiene were aggravated by the problem of migrant labour, where the bread-winner was often forced to seek employment in the towns, leaving his wife and family to fend for themselves. Skeletal tuberculosis was very common and occurred in 49·3 per cent of all orthopaedic patients attending out-patient clinics. The spine was affected in 57 per cent of the total number of patients with skeletal tuberculosis of whom 50·9 per cent were under the age of twelve. Treatment was complicated by inadequate facilities, and admission to hospital was limited to those patients who required immediate surgery. The others were treated as out-patients with isonicotinic hydrochloride, and the affected part was immobilised in plaster. Follow-up proved difficult, for many patients did not report back for re-examination and continuation of treatment. The domiciliary method of treatment—which would be an improvement—was not practicable because of shortage of trained staff. Even so, many lesions were controlled, if not cured. To overcome this complicated problem there must be an improvement in the standard of living of the Bantu and education of ignorant communities by talks and films, and the widespread use of immunisation after mass radiography.

The treatment of the calcaneus foot—*Mr F. J. Hedden* (Durban) said that one of the most disabling types of deformity from poliomyelitis in the growing child was the calcaneus foot. Severe deformity leading to a bad gait resulted when the calf was either completely or partially paralysed, particularly in the presence of a strong tibialis anterior muscle. The calcaneum was rotated in its long axis so that it lay in a more vertical position, and the tuber-calcaneal angle was increased from its normal range of 20–45 degrees to 45–70 degrees. In addition the foot was liable to develop either an eversion or inversion deformity. Treatment was directed to prevention of the development of deformity, and in children under the age of ten it was recommended that tendon transfer to reinforce the

calf should be performed as early as possible, combined when necessary with a Steindler operation if severe cavus deformity had already developed. In a certain number of cases in this age group where there was already a severe valgus deformity, tendon transfer was combined with the introduction of a bone block in the subtalar joint as advocated by Grice. In a number of children in whom a strong tibialis anterior was the only muscle functioning in the foot it was transferred through the interosseous membrane into the calcaneal tendon. In children over the age of ten the Elmslie operation was done in two stages. The cavus deformity was first corrected by a Steindler stripping operation and the talo-navicular joint was fused, and the foot immobilised in full dorsiflexion in plaster for six weeks. At the second stage a wedge of bone was removed from the subtalar joint to correct the deformity and to restore the normal tuber-calcaneal angle, with further immobilisation in plaster for a total of twelve weeks, and this was followed by active physiotherapy. This procedure had given consistently satisfactory results, and many patients were able to stand on tip-toe and had a well shaped foot. Osteotomy of the calcaneum—as described by McFarland of Liverpool—had been done in some children, but the results had been disappointing, and it was felt that in principle it was not a satisfactory operation, as the essential weakness of the calf was left untreated. Moreover, it was found that, in time, the anterior bone blocks had been absorbed and the operation had fallen into disrepute, particularly in the younger children.

Miss Maud Forrester-Brown (Edinburgh) commented on the excellent post-operative appearance of the feet, and agreed that early tendon transfer was essential to prevent development of deformity. In her experience the reinforced calf was seldom sufficiently strong to allow walking on tip-toe. *Mr C. T. Moller* (Johannesburg) had found that the reversed Lambrinudi operation gave satisfactory results in older patients in whom there was marked deformity.

The reduction of frequency and severity of injury in car accidents—*Mr G. T. du Toit* (Johannesburg) said that in South Africa in 1958 there were 101,902 accidents with 2,632 deaths, which cost the country about £30,000,000. The monster of mechanical trauma had been let loose and too little attention was being paid to car design. We were still driving around at murderous speeds in chromium plated juke boxes, whose design bore little relationship to safety. Injuries to the head and face were the most common, followed by those to the legs, chest and thoracic spine. At Cornell University, U.S.A., it had been shown that, of the total number of persons killed, 47 per cent had been ejected from the car. Other injuries were caused by striking the steering wheel and column or rigid objects, such as the instrument panel inside the car. Whip-lash injuries to the cervical spine were all too common, particularly in rear-end collisions. Mathematical calculations carried out at Cornell University showed that the safety of the occupants depended on their decelerating at approximately the same rate as the car itself, and that living humans could be decelerated far more safely than was at first thought possible. If a car was stopped instantly at 30 m.p.h., all the occupants of the car would be killed unless they were restrained from continuing to travel forwards after the car had stopped. It had been shown that safety belts reduced the severe or fatal grades of injury by 80 per cent as compared with non-users of belts, and that there was 85 per cent less risk of dangerous or fatal injury than that experienced by ejected non-users. Padding of instrument panels, collapsible steering wheels, and the use of protective headwear were all found to be of assistance, but the most important single factor was the safety belt. Emergency care of the victims of road accidents was often inadequate, and many hospitals were deficient in their organisation, particularly with regard to early treatment, and considerable delay was often experienced in getting the patient to the operation theatre. More rigorous medical examination of drivers—with particular emphasis on eyesight—and the danger of the abuse of alcohol when driving, were emphasised. It was felt that the Medical Association of South Africa should appoint a committee on trauma to study the problems of prevention and treatment of road accidents.

CLINICAL MEETING

A clinical meeting was held at the Orthopaedic Department, Frere Hospital, East London, on Wednesday, September 30, 1959, under the chairmanship of *Mr L. Mirkin* (Port Elizabeth).

Bilateral congenital dislocation of the hips—*Mr J. Vogel* (East London) showed a patient, aged forty, with bilateral congenital dislocation of the hips which were painful, but the spine was normal. No treatment was recommended.

Multiple epiphysial dysplasia—*Mr J. Vogel* (East London) also showed two children with this condition affecting both hip joints and both lower ends of the tibiae. *Mr G. T. du Toit* (Johannesburg) suggested the use of thyroid extract, but recommended no specific treatment for the involved joints.

Bone grafting—*Dr J. Parsons* (East London) showed radiographs of patients who had undergone various bone-grafting procedures in which the bone had been preserved in ether for a varying period. These cases included grafting for non-union of a fractured tibia, ischio-femoral arthrodesis, excision

of a benign tumour of the tibia, and non-union of the ulna. In the discussion *Mr Percy-Lancaster* mentioned experimental work on rabbits which had demonstrated that ether destroys all organisms—except spore-bearing ones—within a few minutes. Fifty bone grafts by this method had been done in East London and there had been no infection, and the results were so far very promising.

Flexion deformities of the hip—*Dr J. Parsons* showed a number of patients with flexion abduction deformities of the hip treated by Campbell's modification of Soutter's muscle slide operation followed by the use of a special frame developed at the Frere Hospital, which, although dependent on the Dame Agnes Hunt method of correction, allowed a much greater degree of correction to take place.

Recurrent posterior dislocation of the shoulder—*Mr R. Percy-Lancaster* (East London) showed a middle-aged woman who suffered from a recurrent posterior dislocation of the shoulder joint due to excessive spasm of the adductors of the shoulder resulting from encephalitis contracted in infancy. Reefing of the posterior capsule had failed to relieve the condition, but a successful result was eventually obtained by a posterior bone graft.

Bilateral slipped upper femoral epiphyses—*Mr Percy-Lancaster* (East London) showed two patients with bilateral slipped upper femoral epiphyses, one being a boy with clinical features of a patient with hypopituitarism, and the other a girl who was tall and thin. The latter had a fever for a week on each occasion before the slipping developed, and for some weeks subsequently. Both patients had been treated by fixation with Austin Moore pins with excellent reduction, but in the girl one hip developed avascular necrosis. The other three hips were all very satisfactory.

Calcinosis universalis—*Dr E. Sheard* presented a patient with calcinosis universalis, and also a patient who, among other serious injuries, had had a compound fracture of the right clavicle, with division of the supraclavicular nerve. Non-union of the clavicle resulted, and in spite of splintage and physiotherapy active abduction had been impossible until union had been obtained in the clavicle by a bone graft.

ANNOUNCEMENTS

BRITISH ORTHOPAEDIC ASSOCIATION

ARRANGEMENTS FOR MEETINGS IN 1961

The Spring meeting of the British Orthopaedic Association will be held in Manchester from April 13 to 15. Those who wish to submit papers for possible inclusion in the programme for the Spring meeting are asked to send a précis of about 400 words to the Honorary Secretary, British Orthopaedic Association, 47 Lincoln's Inn Fields, London, W.C.2. The closing date for receipt of papers will be December 31, 1960.

The Autumn meeting will be held in London from September 28–30, 1961.

LOVELACE CLINIC, NEW MEXICO

U.S.A.: The Lovelace Clinic, situated in the South West, is expanding its staff of fifty-two physicians. As a result of excellent response to previous advertisement, further applications for full-time appointments are invited in the field of orthopaedics. Opportunities for research are available. Conditions of appointment and detailed information available from the Board of Governors, Lovelace Clinic, Albuquerque, New Mexico.

Book Reviews

Orthopaedics: Principles and their Application. By Samuel L. TUREK, M.D., Attending Orthopaedic Surgeon, Weiss Memorial Hospital; Associate Attending Orthopaedic Surgeon, Mount Sinai Hospital; Alternate Attending Surgeon, Department of Fractures and Traumatic Surgery, Cook County Hospital; Instructor, Department of Orthopaedic Surgery, Northwestern University Medical School. 10×8 in. Pp. xix+906, with 547 figures and 53 colour plates. Index. 1959. London: Pitman Medical Publishing Co. Ltd. Price £8.

Samuel Turek conceived the idea of preparing a truly comprehensive reference book dealing with the principles of orthopaedic surgery. The book was born of a desire to compile scientifically accurate information relating to orthopaedic surgery and to formulate a method by which the facts would be readily accessible. He has not been discouraged by the prospect of arduous labour, and for twenty years he has maintained classified files of information. For the past few years he has sacrificed his own private practice and has given up his leisure time to the preparation of this volume. A work containing so much dedicated effort deserves our respect. We must accept at the outset that this is a book for reference and not a text-book for reading. A text-book is a personal tribute by an author to his own knowledge and opinion based on experience: the information to be had from this book is gleaned from many workers, and Turek carefully avoids opinion, his own or anyone else's, for the sake of recorded facts. Students of orthopaedics in the undergraduate sense will find too much for their digestion, but the graduated orthopaedist will save himself hours of searching through journals and monographs, because the information is all there between two covers and, by the arrangement and indexing of the subject matter, it is easy to find. The masses of illustration and the double column arrangement of print make steady and prolonged reading rather trying, but as a book for quick reference it is unequalled in past or present orthopaedics.

There are four parts: Basic Sciences; General Orthopaedic Conditions; Regional Orthopaedic Conditions; and Special Subjects of which there are only two—Radio-active Isotopes in Orthopaedics and Amputation. Fractures and other injuries of the limbs and trunk are omitted, except in a comprehensive account of conditions affecting the Hand, Dislocation of the Shoulder and Internal Derangement of the Knee. No doubt Turek feels that the book is large enough without including injuries, and does not subscribe to the view that trauma is no longer a part of orthopaedics. There is an extensive section on Peripheral Vascular Disease which is of great value for reference and practical use by those orthopaedists who have no expert in this subject to assist them. There is a good deal of surgical anatomy with beautiful coloured illustrations from many different sources, but those for whom the book will be of real value might regard such inclusions as somewhat naïve; they might consider that space, time and expense could have been saved by their omission. On the whole, operative surgical procedures are unevenly dealt with, and while several different operations may be mentioned the reasons for preferring one operation to another are not made clear. This again perhaps emphasises the function of the book as a ready reference for the experienced surgeon rather than as a guide to the student. In such a dedicated and comprehensive book any attempt at detailed criticism by chapter or section would be invidious, and it is for the users of the volume to find out the rare omissions and inaccuracies.

This is a beautifully produced work and Turek is to be congratulated on his choice of the J. B. Lippincott Company as his publishers. In his preface, which is not the least important part of his book, Samuel Turek pays a tribute to the courage displayed by his wife and family in the face of social restriction and paternal absenteeism imposed by the demands of writing, and he hopes to be restored to the position of father and husband in good standing. His family may rest content that he returns to them with his professional standing assured and elevated by his work.—W. D. COLTART.

Metals and Engineering in Bone and Joint Surgery. By Charles Orville BECHTOL, M.D., Professor of Orthopedic Surgery and Chairman of Orthopedic Division, University of California, Los Angeles; Chairman of Subcommittee on Testing, Prosthetics Research Board, National Research Council; Albert Barnett FERGUSON, Jun., M.D., Silver Professor of Orthopedic Surgery, and Chairman of Orthopedic Department, University of Pittsburgh; Children's and Presbyterian Hospital, Pittsburgh; and Patrick Gowans LAING, M.B., B.S., F.R.C.S., Assistant Professor of Orthopedic Surgery, University of Pittsburgh; Chief of Orthopedic Service, U.S. Veterans' Hospital, Oakland, Pittsburgh. 10×7 in. Pp. vii+186, with 119 figures. Index. 1959. London: Baillière, Tindall & Cox, Ltd. Price £3, 4s.

This book will be appreciated by students of orthopaedic surgery because it brings together within a small compass the published work related to the use of metals and engineering techniques in internal fixations used in orthopaedic surgery. The last chapter by Charles Bechtol is particularly interesting as it describes some original experiments on animals, among which are the effects of screws in weakening tubular long bones.

The chapters on Metal Behaviour and Available Metals deal with purely engineering and metallurgical matters and, though some of it makes heavy reading, will be a valuable source of reference for the composition and properties of various alloys.

I was disappointed that no critical appraisal of the implications of metallic transfer was attempted. Electrolytic action between the under surface of the head of a screw and the plate must undoubtedly be of great importance, but one feels that some other aspects of metallic pick-up as, for instance, that occurring inside the screwdriver slot in the head of a screw, as reported by Bowden, Williamson and Laing (*Journal of Bone and Joint Surgery*, 1955, **37-B**, 676), may have no real practical importance in surgery. The electrolytic witch-hunt must not distract our attention from the failure of the osteoblast to do what we would like it to do at a site several centimetres away from the electrolytic cell.

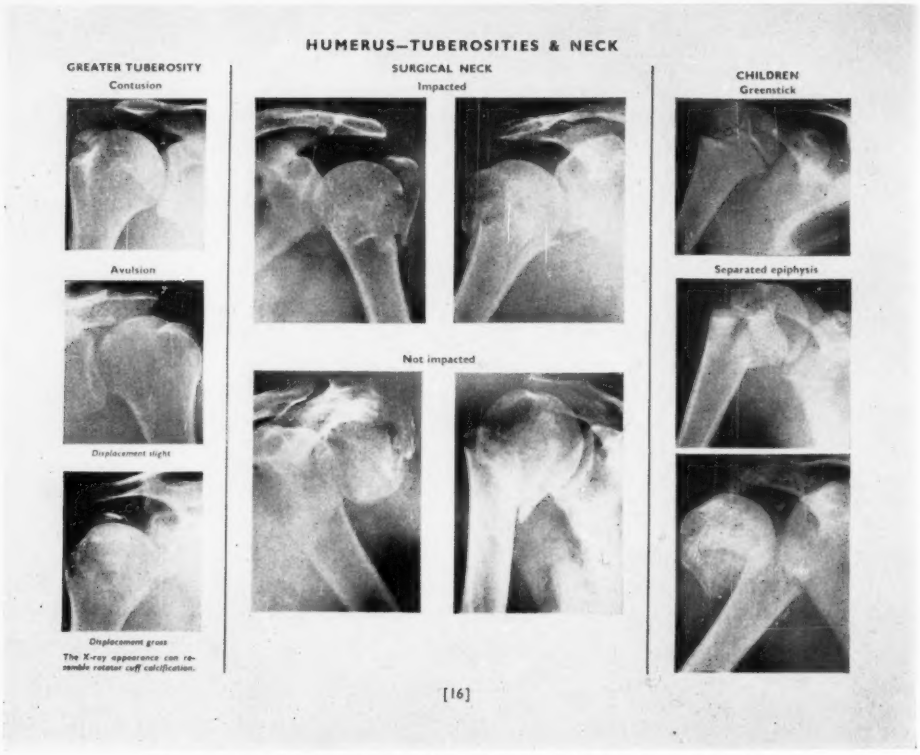
This book is assured of success and doubtless will be followed by others of a similar kind introducing the elements of engineering to orthopaedic surgeons.—John CHARNLEY.

The Care of Minor Hand Injuries. By Adrian E. FLATT, M.A., M.D., F.R.C.S., Assistant Professor of Orthopaedic Surgery, State University of Iowa. With a foreword by Carroll B. LARSON, M.D. 10×7 in. Pp. 266, with 109 figures. Index. 1959. St Louis: The C. V. Mosby Company. London: Henry Kimpton. Price 71s. 6d.

The author has an original approach to his subject; his book is out of the ordinary and for that reason is more attractive than most. It makes interesting and informative reading, valuable both for surgeons who are inexperienced and for those who will use it for reference.

The book is divided into two parts: the first deals with the common-denominator knowledge that is applied throughout hand surgery; and the second deals with particular injuries and their remedy. Some repetition is unavoidable since the book is meant to be referred to as occasion arises. When several rival methods of treatment are in vogue for a particular injury the author recounts the system that he has found from experience to be both practical and successful. For all that the reader is not likely to be impressed by the suggestion that the fragments of a Bennett's fracture can be held in place by sutures through the periosteum. Nor is the reader likely to believe that " a fracture of the neck of a metacarpal is a serious injury " because dorsal angulation at the fracture line " produces such an imbalance of the extrinsic and intrinsic muscles of the finger that function would be severely limited if the fracture united in an unreduced position." Some surgeons believe that a fracture of the metacarpal neck is best treated by active movement and that the usual volar displacement of the metacarpal head does not matter very much and is not ordinarily associated with later disability. At least that is what I think. Slight differences of opinion are likely to be a matter of degree but even these could be cleared up if there were more radiographs so that the reader knew exactly what the author had in mind. The section on soft-tissue injury as it is dealt with in the casualty department is excellently done, and no casualty officer could fail to profit from an intensive study of these pages.

The book is well produced, the illustrations, both photographs and line drawings, are excellent, and in nearly all instances correspond with the text. The author is to be congratulated on writing such an interesting and readable volume which will take its place among the handbooks on hand surgery.—R. FURLONG.



Specimen page (reduced) from Wiles: *Fractures, Dislocations and Sprains*

Fractures, Dislocations and Sprains. By Philip WILES, M.S.(Lond.), F.R.C.S.(Eng.), F.A.C.S., Honorary Consultant Orthopaedic Surgeon, the Middlesex Hospital. *Past President*, the British Orthopaedic Association; the Orthopaedic Section, the Royal Society of Medicine; and the Orthopaedic Section, the British Medical Association. Corresponding Member, the American Orthopaedic Association. *Formerly* Hunterian Professor, the Royal College of Surgeons of England; Lecturer in Orthopaedic Surgery, the University of London; Brigadier, Consultant Surgeon in the Army. British Treasurer, *The Journal of Bone and Joint Surgery*. 10×12½ in. Pp. 67, with 519 figures. 1960. London: J. & A. Churchill Ltd. Price 27s. 6d.

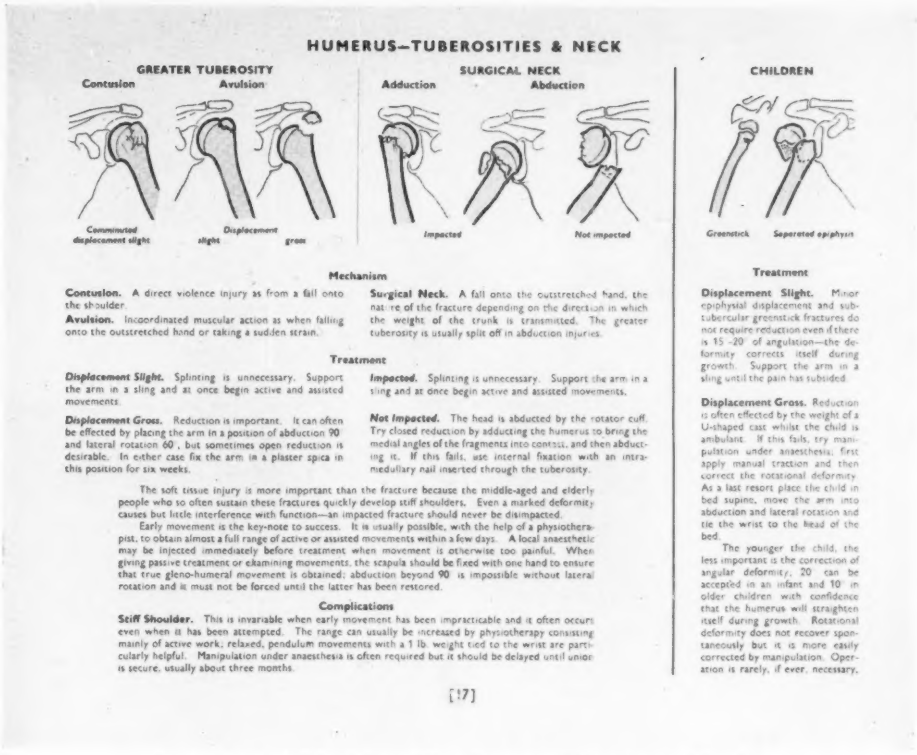
In the preface to his book Philip Wiles states that his intention has been to produce a short, generously illustrated volume chiefly for the use of casualty officers, house surgeons and students, supplying in a succinct form the information necessary for the diagnosis and treatment of fractures and dislocations.

He has done just this and done it splendidly.

With the present organisation of accident and casualty departments in this country, comparatively recently qualified casualty officers and house surgeons are faced with the responsibility of the primary treatment of many fractures. These harassed and overworked young doctors have not the time or the energy to refer to the larger text-books. What they want is a quick and easy reference book which is practical and safe.' This is it.

The book is so arranged that each anatomical region is covered by two facing pages. With this format the precise nature of most injuries and the problems to which they may give rise, together with the appropriate treatment, can be identified quickly and easily.

The author wisely omits any description of the operative technique of open reduction or internal fixation, but describes clearly and simply the methods of manipulative reduction and external fixation of simple fractures and dislocations. Thus the inexperienced young doctor is easily able to distinguish the injuries he can treat from those he must refer to a more experienced surgeon.



HUMERUS—TUBEROSITIES & NECK

GREATER TUBEROSITY

Contusion Avulsion

Comminuted displacement slight

Displacement slight great

SURGICAL NECK

Adduction Abduction

Impacted

Not impacted

CHILDREN

Greenstick

Separated epiphysis

Mechanism

Contusion. A direct violence injury as from a fall onto the shoulder.

Avulsion. Incoordinated muscular action as when falling onto the outstretched hand or taking a sudden strain.

Surgical Neck. A fall onto the outstretched hand, the nature of the fracture depending on the direction in which the weight of the trunk is transmitted. The greater tuberosity is usually split off in abduction injuries.

Treatment

Displacement Slight. Splinting is unnecessary. Support the arm in a sling and at once begin active and assisted movements.

Displacement Gross. Reduction is important. It can often be effected by placing the arm in a position of abduction 90° and lateral rotation 60°, but sometimes open reduction is desirable. In either case fix the arm in a plaster spica in this position for six weeks.

Impacted. Splinting is unnecessary. Support the arm in a sling and at once begin active and assisted movements.

Not Impacted. The head is abducted by the rotator cuff. Try closed reduction by adducting the humerus to bring the medial angles of the fragments into contact, and then abducting it. If this fails, use internal fixation with an intramedullary nail inserted through the tuberosity.

The soft tissue injury is more important than the fracture because the middle-aged and elderly people who so often sustain these fractures quickly develop stiff shoulders. Even a marked deformity causes but little interference with function—an impacted fracture should never be disimpacted.

Early movement is the key-note to success. It is usually possible, with the help of a physiotherapist, to obtain almost a full range of active or assisted movements within a few days. A local anaesthetic may be injected immediately before treatment when movement is otherwise too painful. When giving passive treatment or examining movements, the scapula should be fixed with one hand to ensure that true gleno-humeral movement is obtained; abduction beyond 90° is impossible without lateral rotation and it must not be forced until the latter has been restored.

Complications

Stiff Shoulder. This is invariable when early movement has been impracticable and it often occurs even when it has been attempted. The range can usually be increased by physiotherapy consisting mainly of active work; relaxed, pendulum movements with a 1 lb. weight tied to the wrist are particularly helpful. Manipulation under anaesthesia is often required but it should be delayed until union is secure, usually about three months.

Treatment

Displacement Slight. Minor epiphysial displacement and subtubercular greenstick fractures do not require reduction even if there is 15°–20° of angulation—the deformity corrects itself during growth. Support the arm in a sling until the pain has subsided.

Displacement Gross. Reduction is often effected by the weight of a U-shaped cast whilst the child is ambulant. If this fails, try manipulation under anaesthesia; first apply manual traction and then correct the rotational deformity. As a last resort place the child in bed supine, move the arm into abduction and lateral rotation and tie the wrist to the head of the bed.

The younger the child, the less important is the correction of angular deformity; 20° can be accepted in an infant and 10° in older children with confidence that the humerus will straighten itself during growth. Rotational deformity does not recover spontaneously but it is more easily corrected by manipulation. Operation is rarely, if ever, necessary.

[17]

Specimen page (reduced) from Wiles: *Fractures, Dislocations and Sprains*

A book of this type is necessarily dogmatic and because of this some experienced orthopaedic surgeons might find in it much to criticise. I found very little and that unimportant, for my bias, in common with the author, is towards a conservative rather than an operative approach.

A dogmatic approach is often necessary when a master is instructing pupils. Philip Wiles is certainly a master and his pupils extend more widely than the junior house officers to whom his book is primarily addressed.

I recommend this book to all surgeons engaged in the treatment of fractures, and to all hospital management committees. They would do well to see that there is at least one copy in all casualty departments.—F. W. HOLDSWORTH.

Modern Trends in Accident Surgery and Medicine. Edited by Ruscoe CLARKE, *M.B.E.*, M.B., F.R.C.S.(Eng.); F. G. BADGER, B.Sc., F.R.C.S.(Ed.); and Simon SEVITT, M.D., M.Sc., F.R.C.P.I., D.P.H., of the Birmingham Accident Hospital, with seven other contributors. 10×7½ in. Pp. xi+346, with 82 figures. Index. 1959. London: Butterworth & Co. (Publishers) Ltd. Price 75s.

This latest addition to the " Modern Trends " series is the work of a number of contributors who either are, or have been, members of the staff of the Birmingham Accident Hospital—and the first thing about it is its title. What, one might ask, is " Accident Medicine "? The answer, to quote from the preface, is that " a modern understanding of accident cases involves the patient as a whole, that is the medical, physiological, pathological as well as the surgical aspects." Under the general editorship of two surgeons and a pathologist " accident medicine " does, in fact, occupy precisely one-third of the entire volume. There are excellent chapters on systemic reactions to injury (cardiovascular, nervous, endocrine and metabolic), the haemodynamics of blood loss, traumatic uraemia, fat embolism and thrombo-embolism, the latter dealing particularly with Sevitt's recent

work on prophylactic anticoagulant therapy. In short " accident medicine " is one of the best features of the book and will amply reward the busy orthopaedic surgeon who has allowed himself to become " rusty " in physiology and pathology.

Throughout the work one sees the inspiration of Ruscoe Clarke whose death so sadly occurred just after it was published. In particular he dealt with resuscitation and transfusion and argued the case for immediate and rapid replacement of the whole amount of blood lost whenever this exceeds 1½ pints, even in fit young adults. Since most closed fractures of the femur are stated to lose from 1 to 4 pints of blood into the tissues, this policy would appear to advocate a more liberal use of blood transfusion than is at present practised in this country.

Badger writes on the treatment of fractures. In an excellent introduction dealing with general principles and bearing the hallmark of great practical experience, he notes the current trend towards more internal fixation, but wisely remarks that " those fractures which will unite in reasonable time, without significant disability when treated by closed methods, should still be so treated." Not everyone will agree with his advocacy of immediate plating in open fractures, or be reassured by his statement that should the plate become exposed by the sloughing of traumatised skin " suitable dressings can prevent infection and the exposed plate will be covered over by granulation tissue and the spread of epithelium." In these days of direct violence the skin overlying a fractured tibia is particularly vulnerable and the reappearance of a plate from sloughing of tissues is more often the prelude to a depressingly familiar chain of events, including infection, multiple sequestrectomies, pedicle grafting, non-union, failed bone grafting and, not infrequently, amputation about two years later. This reverse side of the picture deserves more emphasis in a condition which can be treated by safe and simple methods with excellent results.

The chapter that is perhaps the most controversial is written by J. H. Hicks on the internal fixation of fractures. His thesis is that fixation, to be effective, must be absolute. (Twenty years ago it was anatomical reduction that had to be absolute.) He emphasises that plaster never fixes a fracture and that current methods of internal fixation are but an approximation. He claims that both joint stiffness and delayed union arise from imperfect fixation, and that when the perfect plate has been invented (the Hicks model is " promising but it is still too early to recommend it for general use ") fractures will unite without visible callus and joints will no longer become stiff because they will be released from the inhibiting effect of five degrees of movement at the site of fracture. He adds that the periosteum must not be stripped, open fractures should be plated even when grossly contaminated, and the plate should never be removed if infection supervenes. These forthright views are advanced with admirable verve and assurance, in the course of which Watson-Jones is dismissed as a fatalist and Charnley as an infidel.

Now that the general surgeon has abdicated from the control of accident services, it is in the realm of head, chest and abdominal injuries that most danger exists. These are the *killer* injuries and the patient must not be allowed to fall between two stools. The orthopaedic surgeon must know enough to recognise the indications for consulting his colleagues in other departments or, in the more remote areas, be able to do himself what is immediately necessary as a life-saving measure. These problems are dealt with in an excellent chapter on abdominal and chest injuries and attention is directed once again to that lethal condition known as " flail chest " in which early tracheotomy and stabilisation of the floating rib segment may save life dramatically. Similar guidance on the management of serious head injuries and the indications for surgical interference would have been a welcome addition to this section.

In their preface the editors state that their chief difficulty has been to decide what to leave out. If one had to criticise their selection it would be that certain controversial topics of current interest have been omitted; whereas other topics which have become stereotyped and about which there has been universal agreement for many years have been included. In the former category one thinks of intracapsular fractures of the neck of the femur with its spate of new gadgets, fractures of the carpal scaphoid, and fractures of the spine particularly when associated with paraplegia, to name only a few of the topics that can still divide any meeting of orthopaedic surgeons.

This is a book that should be read by every surgeon responsible for the treatment of injuries. It does not pretend to reflect contemporary thought and practice; indeed, it is frankly individualistic, didactic, controversial and even provocative. This makes it all the more stimulating and enjoyable. Experienced surgeons will disagree with much of it, particularly if they come from the " non-plating zone " in this country which starts just north of Birmingham. That is no doubt what the authors expect. But the sincere views of men with original minds and considerable experience must always command respect, and any book on this notoriously controversial subject that failed to generate a little heat would not be worth reading. My favourite cynic is always reminding me that in the best book on fractures ever to be written in any language, there is a lie on every page.—E. A. NICOLL.

The Surgical Treatment of Scoliosis. By Louis A. GOLDSTEIN, M.D., F.A.C.S., Associate Clinical Professor of Orthopaedic Surgery, University of Rochester Medical Center; Associate Orthopaedic Surgeon, Strong Memorial Hospital of the University of Rochester Medical Center. Appendix **Anesthesia in Scoliosis.** By D. Vernon THOMAS, M.B., F.F.A.R.C.S., Professor of Anesthesia, Anesthetist-in-Chief and Head of the Division of Anesthesia, Strong Memorial Hospital of the University of Rochester Medical Center. 10×7 in. Pp. xii+116, with 47 figures. Index. 1959. Springfield, Illinois: Charles C. Thomas, Publisher. Oxford: Blackwell Scientific Publications Ltd. Toronto: The Ryerson Press. Price 54s.

This monograph describes one surgeon's experience in the treatment of structural scoliosis during twenty years. Dr Goldstein describes his methods of conservative treatment, consisting essentially of careful observation with postural exercises and exercises designed to preserve spinal mobility. The treatment of paralytic deformity by prolonged recumbency, stretching exercises, bent plaster jackets and the Milwaukee brace is discussed, and mention is made of the elimination of deforming contractures such as those of the ilio-tibial band.

Sixty-five patients who were treated by spinal fusion are described, fifty-two of whom had been followed up for two years or more and two for eighteen months. The methods of correction and fusion are devolved from those of Cobb, Risser and Ferguson. The indications for operation are: serious and increasing deformity before the end of spinal growth, ugly correctible deformity at or after maturity, and, in two adults, pain. The extent of fusion comprises the primary curve or curves; and correction is obtained in the Risser turnbuckle plaster jacket, the technique of application being well described. Radiographs are taken with the patient bending sideways in the direction of the concavity of the primary curve in order to determine the fixed deformity in the compensatory curves, the aim being to avoid correction of the primary curve beyond the sum of the secondary curves. Two cases in which overcorrection took place are illustrated. The operation for spinal fusion is described in detail. It consists in a wide subperiosteal exposure followed by decortication with hand gouges and the application of large quantities of supplementary bone. This was obtained from the patient's tibia between 1939 and 1947, a bone bank between 1948 and 1951, and the patient's ilium since 1952. After operation the patients are recumbent in plaster for eight months. This period of recumbency is spent mostly in their own homes, where education is continued, and is followed by two months in a walking plaster cast. In 75 per cent of the patients the curves before operation were over 70 degrees. Loss of 15–24 per cent of correction was considered possible from bending of the graft. The incidence of pseudarthrosis varied with the causation of the curve, and was 13 per cent in the fifty-four cases followed up, being significantly lower since 1952 when autogenous bone was used as the supplement.

Dr Goldstein is fortunate in having had throughout one anaesthetist in the person of Professor Vernon Thomas, who writes a final chapter. All patients had endotracheal intubation; the head-piece of the plaster was not disturbed but a large window was cut before operation to allow access to the trachea and chest in an emergency. Two tracheotomies were performed in this series. There were no deaths.

The illustrations are very good throughout. They show a considerable number of excellent results as well as a very fair proportion of the less successful. The poor results are discussed in detail.—Charles MANNING.

Die Wirbelsäulenleiden und ihre Differentialdiagnose. By Prof. Dr J. E. W. BROCHER, Genf. Enlarged second edition. 11¼×8½ in. Pp. xv+457, with 639 illustrations in 287 figures. Index. 1959. Stuttgart: Georg Thieme Verlag. Price DM 128.

Any book that sheds fresh light on the difficult subject of backache—its causes, differential diagnosis and treatment—is sure of a welcome reception by the orthopaedic surgeon. The volume now under review is undoubtedly a valuable contribution to the better understanding and therefore the more rational treatment of this all-too-frequent complaint common to all spinal lesions. Dr Brocher has spared no pains in the preparation of his book and has given us the results of many years of work in this field together with a critical examination of the work of others. He lays his main emphasis on the differential diagnosis of spinal lesions, and with this end in view he begins by giving a very careful and detailed description of the signs and symptoms and particularly the radiographic appearances of each condition. He emphasises the value of tomography and also the necessity of including in radiographs a large section and often all regions of the spine, and sometimes the shoulder and pelvic girdles as well. He recommends that films be taken in the erect posture at a distance of 150 centimetres in order to reveal any deviation from the normal axis of the vertebral column. Skilled radiography and experience and training in reading the films are of course essential.

Treatments are given in broad outlines only. The various orthodox methods are briefly discussed, a conservative attitude to surgical intervention is advised, and no new forms of treatment are recommended.

The book is divided into fourteen sections including a short epilogue. The first section, devoted to tuberculosis of the spine, occupies 105 pages, nearly a quarter of the whole book, whereas all degenerative lesions including prolapsed intervertebral discs occupy only thirty-nine pages, and traumatic lesions are not described at all, being mentioned only in relation to the differential diagnosis of other conditions. In his preface the author justifies his eclecticism on the ground that these other conditions are so fully treated elsewhere. While the skill and detailed care in the description of tuberculosis of the spine are appreciated, it seems a pity that a fortunately decreasing malady should occupy so disproportionately large a part of this book. The third section gives a very full account of spondylitis ankylopoetica and the need for early diagnosis, but a discussion of etiology at some length brings nothing new to our notice. A most interesting fourth section deals with Scheuermann's disease on which the author has several new theories which he sets forth with clarity and plausibility. He rates the importance of this disease very high, because he dates the onset as early as the eighth or ninth year and because, although causing little trouble in childhood, the disease may in adult life be a not infrequent cause of brachial neuritis, scalenus anterior syndrome, lumbago and sciatica from late failure of the compensating mechanism between bone structure and musculature.

Other sections give very good descriptions of Calvé's disease, congenital abnormalities, degenerative lesions, neoplasms, and such conditions as osteoporosis and osteosclerosis of the spinal column; and there is a very useful section on the pathology of the sacrum. Throughout, the importance of the intervertebral disc and its changes in height or herniation into the body of a neighbouring vertebra are described with great care and well illustrated by radiographs.

The last section is devoted to the differential diagnosis of spinal lesions and is concerned almost exclusively with the radiographic evidence. The changes that may be found in the intervertebral discs are discussed first, followed by a most careful consideration of the pathology of each part of the vertebra in turn. In this way the characteristic radiographic appearances of all the spinal diseases are distinguished from one another, apparent similarities which might lead to an error of diagnosis are explained, and small changes that might easily be missed are emphasised. It is perhaps the most rewarding of all the sections. The excellence and careful interpretation of the radiographs are above praise, and consequently the book may well prove as valuable to the radiologist as to the orthopaedic surgeon.

In the epilogue Dr Brocher reaffirms the importance he attaches to radiography for the early detection of spinal disease and the differential diagnosis.

The book is beautifully produced and lavishly illustrated. At the end there is an extensive alphabetically arranged bibliography for each section. There is also a good index.—E. Henrietta JEBENS.

Principles of Bone X-ray Diagnosis. By George SIMON, M.D., M.R.C.P., F.F.R., Assistant Director, X-ray Department, Brompton Hospital, London; Radiologist, St Bartholomew's Hospital, London; Demonstrator, Radiological Anatomy, St Bartholomew's Hospital, London; Curator of the Radiological Museum and Teacher of Radiology, The Institute of Diseases of the Chest, University of London. 11 × 8¾ in. Pp. xxi + 178, with 244 figures and 7 tables. 1960. London: Butterworth & Co. (Publishers) Ltd. Price 57s. 6d.

The idea behind this book is attractive and the execution creditable, but the final result is nevertheless not entirely satisfactory. The author has split the book up into chapters under such headings as alterations in bone shape, abnormalities in the region of the epiphyses, increases of bone density, new bone and calcification outside the cortex, a translucent area in bone and so on; and his trouble derives from the fact that the changes which a bone can undergo radiologically, while limited in number and type, are almost always seen not in isolation but in a variety of combinations. Consequently, there is too much duplication and repetition from chapter to chapter, and many of the illustrations relevant to a particular paragraph are sited many pages away, which makes the book uncomfortable to read. There appear to be one or two misdiagnoses in the excellently reproduced radiographs; and the clinical paragraphs, particularly from the orthopaedic point of view, are not all as good as the rest of the book: if a second edition were required the author would be well advised to seek the collaboration of an orthopaedic colleague to bring these paragraphs into line with modern thought.

As part of the education of the budding radiologist this book will fill a useful place, and certainly the young orthopaedic surgeon will find much to interest and instruct him. In the long run, however, interpretation of difficult radiographs is a matter of experience; so subtle are the individual changes

that it is only by looking at the picture as a whole that the expert can make a diagnosis, and even he may have difficulty in pointing out the exact factors on which his decision is reached. The position is comparable to that of the countryman who can identify a distant bird without hesitation and yet have great difficulty in explaining just how or why he knows the bird to be what it is.—T. J. FAIRBANK.

Radiology as a Diagnostic Aid in Clinical Surgery. By Howard MIDDLEMISS, M.D., F.F.R., D.M.R.D., Director of Radiology, United Bristol Hospitals; Lecturer-in-Charge, Department of Radiodiagnosis, University of Bristol. $9\frac{1}{2} \times 6\frac{1}{2}$ in. Pp. viii + 151, with 86 figures. Index. 1960. London: William Heinemann Medical Books Ltd. Price 30s.

This short book is designed to keep practising surgeons up to date in modern radiological methods and to indicate not only the advantages, but also the limitations, of this method of diagnosis. The author begins by considering the value of emergency and post-operative investigations and then discusses the various anatomical systems as sites of special study. The radiographs are all classical examples of disease processes and are beautifully reproduced. Although the book should be of particular value to the senior student and the newly qualified man every reader will find much to refresh the memory and will almost certainly learn something new. Nevertheless its brevity has meant that, to the surgeon in a particular speciality, much that would appear to be of importance has necessarily been omitted. Such a specialist, including the orthopaedic surgeon, will inevitably find some conflict of opinion on a number of minor points though the major principles are well stated. The chapter on bone radiology represents one-fifth of the book, but those interested in orthopaedics may regret that some subjects of importance, such as spondylolisthesis, have been given scant attention, and some recent observations, such as the differential diagnosis of osteoclastoma from the benign tumours of bone, receive virtually no reference. Nevertheless this book can be read without difficulty in a few hours and the author's easy style makes it a pleasant experience. It should lead to improvement in diagnostic standards from better co-operation between the surgeon and the radiologist.—R. O. MURRAY.

Atlas of Anatomy and Surgical Approaches in Orthopaedic Surgery—Upper Extremity. By Rodolfo COSENTINO, M.D., Assistant Professor in Orthopaedic Surgery, University of La Plata; Research Associate, Department of Orthopaedic Surgery, State University of Iowa. With a Preface by Arthur STEINDLER. $11\frac{1}{4} \times 8\frac{3}{4}$. Pp. xiv + 192, with 134 figures. 1960. Springfield, Illinois: Charles C. Thomas, Publisher. Oxford: Blackwell Scientific Publications Ltd. Price 84s.

This atlas of upper limb anatomy was designed by Dr Cosentino while working in the Department of Orthopaedic Surgery, University of Iowa, and there is a preface by Arthur Steindler. As might be expected from such origins, the production is excellent—especially the illustrations, which are easy to understand. It will be useful for quick reference before an operation if the anatomy is a little unfamiliar to the operator. The present atlas of orthopaedic approaches to the upper limb is to be followed by volumes on the lower limb and the trunk, and we look forward to these.—J. I. P. JAMES.

Rehabilitation Medicine. A Textbook on Physical Medicine and Rehabilitation. By Howard A. RUSK, M.D., Professor and Chairman of the Department of Physical Medicine and Rehabilitation, New York University-Bellevue Medical Center, New York, N.Y. With 36 collaborators, and with the editorial assistance of Eugene J. TAYLOR, A.M. 10×7 in. Pp. 572, with 172 figures and 12 tables. Index. 1958. St Louis: The C. V. Mosby Company. London: Henry Kimpton. Price 90s.

This is an authoritative work—a book to read and also to keep for reference. Throughout, the approach is practical and based on long personal experience at the New York University-Bellevue Medical Center. It is mainly concerned with the rehabilitation of patients with disorders of the limbs, though chapters are included on patients with pulmonary and cardiovascular problems. The emphasis is on treating the whole patient; "patient motivation" and social, psychological and vocational evaluation are recurring themes. Functional capacity and economic potential are considered to be nearly as important as diagnosis in the restricted nosological sense. Stress is laid on the importance of deciding the goals of each type of therapy. The section on self-help devices is outstanding. The authors consider canes, crutches, braces and wheel chairs to be energy conserving devices and not "last resorts." For poliomyelitis they recommend exploitation of the patient's remaining abilities; for cerebral palsy they restrict all but two movements of the affected extremity by a suitable prosthetic appliance. Who could improve on such wisdom? Their *pro forma* for wheel chair prescription should be used in every hospital. Only in advocating laminectomy for traumatic thoracic paraplegia do they differ from current practice in this country. This book would be of value to every physiotherapy department and every orthopaedic surgeon.—Robert ROAF.

Instructional Course Lectures of the American Academy of Orthopaedic Surgeons. Edited by Fred C. REYNOLDS, M.D., St Louis, Missouri. Volume XVI. 11×9 in. Pp. 335, with 260 figures and 36 tables. Index. 1959. St Louis: The C. V. Mosby Company. London: Henry Kimpton. Price £6.

This the sixteenth volume in the series is a selection from the 124 Instructional Courses presented at the 1959 Meeting of the American Academy of Orthopaedic Surgeons. These courses were presumably originally designed for the purpose of keeping busy orthopaedic surgeons abreast of developments in the speciality, but perhaps there is now a tendency to regard them as a substitute for basic reading, at least this is the impression that one gets from this volume.

Some of the articles are pedestrian and would hardly whet the appetite of a jaded surgeon in the early hours of the morning, and one wonders why they were selected in preference to others potentially more interesting that have been excluded. The lecture on the anatomy of the hand adds nothing to the standard texts, and Figure 34, illustrating the sensory nerve supply, is incorrect. The variable distribution of the median and ulnar nerves to the intrinsic muscles of the hand—which is of considerable practical importance—is not mentioned.

There are, however, several excellent articles, especially those by Riordan, on the surgery of the paralytic hand, Grice on the role of subtalar fusion in the treatment of valgus deformities of the foot, and Schmidt on the fundamental principles and treatment of scoliosis. The latter is a delightful and concise presentation of a difficult subject which should be compulsory reading for registrars. These articles alone will make this volume a welcome addition to any library, but even in these days of inflation the price seems a little high.—Roland BARNES.

Biological Problems of Grafting. A Symposium sponsored by the Commission Administrative du Patrimoine Universitaire de Liége, and the Council for International Organizations of Medical Sciences. Established under the joint auspices of Unesco and W.H.O. (18th–21st of March 1959.) Edited by F. ALBERT and P. B. MEDAWAR, with thirty-six other contributors. 10×7 in. Pp. xii+453, with 44 figures and 82 tables. 1959. Oxford: Blackwell Scientific Publications Ltd. Springfield, Illinois: Charles C. Thomas, Publisher. Toronto: The Ryerson Press. Price 50s.

Knowledge of the nature of the reaction between hosts and grafts from individuals of the same species and from other species is accumulating so rapidly that no text-book gives an adequate account of this field. This symposium, while excellent in itself, unfortunately does not fill this gap. Each paper presents an intensive study of some small facet of the whole problem; and, while each is valuable, there is some overlapping and a lack of continuity. A reader not familiar with the subject will find the symposium difficult to follow. Many of the papers are concerned with identification and characterisation of the two types of antibody—humoral and cellbound—that are recognised to develop in response to a homograft: but the relative importance of these, and the mechanisms by which they bring about the rejection of the graft, remain obscure.

Clinicians will perhaps be most interested in those papers that describe methods by which the homograft rejection may be prevented or delayed. The two main techniques at present available—the induction of " tolerance " by exposing an animal to a homograft at or soon after birth, and total body irradiation—while of great interest experimentally, are still unsuited to widespread clinical use. Several papers report that when acceptance of the graft is obtained by one of these means, the host may later sicken and die, because of an immunological reaction of the graft against the host, an observation which will further complicate the therapeutic usefulness of homografts.

Bone grafts receive little attention. One paper by Joubert recommends the clinical use of a calf bone bank on the basis of somewhat slender experimental and clinical evidence and despite the fact that the use of bone heterografts has been found unsatisfactory in other centres, notably in the well documented experience of the Dutch Red Cross Bone Bank. Danis records his experience with grafts of bone marrow, periosteum and fracture callus. Autografts of each of these materials produced a small ossicle containing marrow, whereas homografts failed to do so. From this he concludes that metaplastic induction of bone does not occur and that bone forming around a graft is a product of graft cells—a conclusion with which many would disagree, at least if meant for more general application as he implies.

To those engaged in research in the field of transplantation immunity this book will prove most valuable, as well as indicating many avenues to which their researches could usefully be directed. For the clinician it emphasises how far we have yet to go before living homografts become a reality.—John CHALMERS.

Surgery and Clinical Pathology in the Tropics. By Charles BOWESMAN, *O.B.E.*, M.D., F.R.C.S.E., F.A.C.S., D.T.M.&H.Ed. Formerly Medical Officer, Gambia, West Africa; Surgical Specialist, Gold Coast and Ghana Governments. 10×8 in. Pp. viii+1,068, with 321 figures. Index. 1960. Edinburgh and London: E. & S. Livingstone Ltd. Price £5, 10s.

The author has set himself the task in a most ambitious way of covering the whole field of surgery in the tropics. In the introduction he indicates what little help there is in medical literature for the young surgeon in the tropics who relies on his education from standard text-books of surgery. Although there is much written on tropical medicine, that on surgical problems is sparse and mostly out of date.

Bowesman pays homage to the general principles of surgery and discusses, at times fully, the conditions common both to temperate and tropical climates. Of particular value are his descriptions of the characteristic modifications that occur in work of this sort. There is a chapter of over 200 pages on abdominal surgery which is loaded with observations of this nature. In addition he plunges courageously and profoundly into indigenous problems with the knowledge of one who has great experience in this field. Chapters on sickle cell disease in surgery, helminthic infections, schistosomiasis, ritual operations in the tropics, plastic surgery with special reference to keloid formation and bites of animals and snakes, and the effects of other noxious agents make fascinating reading in this country and will certainly be of great assistance to the tropical surgeon or the general factotum working in isolation.

The surgeon responsible for injuries of the locomotor system and interested in orthopaedic surgery will find the space allotted to these subjects disappointing and poorly representative of the demand for this branch of surgery. He will be made conscious of the possibility of having to treat fractures without radiological control but, finding himself in such a situation, will be assisted little by this book. There is practically no mention of bone and joint tuberculosis nor of the orthopaedic and reconstructive treatment of poliomyelitis and leprosy. To suggest that Nicola's operation for recurrent dislocation of the shoulder is the best available is a reversion to pre-war concepts.

It is not surprising that much has been omitted but astounding that so vast a field has been covered and regarded in such light; this book will make a notable addition to surgical literature in a field which so far is comparatively neglected. Whether it fills and adequately bridges the gap must be debatable. It will have to prove its value as a standard work among the new medical schools of the tropics and run the gauntlet both of praise and criticism of the teachers and students of those pioneer institutions. To the student it has two superficial disadvantages: it is inconveniently heavy and it is costly. Its main value will be as a book of reference and as a companion to surgeons in the tropics.—Philip NEWMAN.

The Effect of Advancing Age upon the Human Spinal Cord. By L. Raymond MORRISON, M.D., with the collaboration of Stanley COBB, M.D., and Walter BAUER, M.D. 11¼×8½ in. Pp. x+127, with 89 figures. 1959. Cambridge, Mass.: Harvard University Press. London: Oxford University Press. Price 48s.

There can be few pathologists who have not longed for a series of volumes in which the range of normal histological appearances of various organs are clearly set out, particular emphasis being laid upon variations with physiological activity and age. Neuropathologists do not differ from their colleagues in this respect and there is no doubt that many will consult this book by Dr Morrison and his collaborators.

The book, which is well produced apart from an occasional untidy and unclear photomicrograph, is divided into two parts. The first part, which deals specifically with the effects of ageing on the spinal cord, was a by-product of a study of the neurological disturbances in rheumatoid arthritis, the investigators having to establish the " normal for age " set of appearances before proceeding with their main investigation. Accordingly the appearances of the cord in infancy and the first decade are not included, which is regrettable. They would have enhanced the value of the book.

Thirty-one spinal cords were studied, the cases being grouped in decades from the second to the ninth. They were obtained from patients dying from non-neurological disease in a general hospital and representative blocks were taken from the cervical, thoracic and lumbar regions. The disease in each case is not listed though there were cases of malignant hypertension, rheumatic carditis, disseminated lupus erythematosus, miliary tuberculosis and carcinomatosis.

It is realised that it takes much time and is often difficult to obtain tissues from cases of sudden death in otherwise healthy subjects of the appropriate age, but it is felt that this work would have been immeasurably improved if this had been done. In presenting the findings " no attempt, or at least very little, was made to correlate the histological findings with the systemic diseases of the patients, for there were too few cases of any one diagnosis in this series to make such a correlation valuable." Because of this, and the failure to discuss the cases individually, the reader is unable

to assess for himself the findings that are a consequence of disease and those that are due to ageing. To some extent these objections are overcome in the discussion and in the summary at the end of the book, but there remains a feeling of regret at the unsatisfactory selection.

Part II is an atlas of a " normal " spinal cord which was obtained shortly after death from a woman of twenty-four dying of subacute bacterial endocarditis. Photographs of Weigert sections and a brief description of the characteristics of each segment of the cord are given, together with photographs of Nissl preparations and an enumeration of the nerve cells in the anterior horns, intermediolateral and Clarke's columns. Considerable care has been taken with this aspect of the study and one cannot help wishing that comparable counts had been done on the material in the first part of the book. The quality of the photomicrographs of the Nissl preparations leaves much to be desired; many are untidy and the definition is often imperfect. This is an interesting book with useful information but can be recommended only with these reservations.—W. BLACKWOOD.

Le Phénomène de Raynaud. Aspects cliniques, étio-pathogéniques et thérapeutiques. By Paul LANGERON and Lucien CROCCEL. $8\frac{1}{2} \times 5\frac{1}{2}$ in. Pp. 198, with 8 figures and several tables. Index. 1960. Paris: L'Expansion Scientifique Française. Price 19.50 NF.

What is Raynaud's disease? This monograph opens with this question, asked so often and never adequately answered. The authors give careful consideration to the phenomena described in Maurice Raynaud's ambiguous original paper and, if they add little that is new, they certainly review all the available literature and clinical and experimental data which have accumulated around this mysterious syndrome. They distinguish between Raynaud's phenomena produced by local factors, by " regional " lesions (in the upper limb, arising mainly in the retroclavicular area), as a response to general diseases and arising apparently idiopathically. They dislike the concept of idiopathic Raynaud's " disease," but are compelled to retain it. The book is short and easily read, and is recommended to any surgeon who wants a handy and authoritative survey of this malady.—D. Ll. GRIFFITHS.

Function Test for Peripheral Arterial Circulatory Insufficiency in the Lower Extremities in Obstructive Arterial Disease. By Björn L. LINDSTRÖM. $9\frac{1}{2} \times 7\frac{1}{4}$ in. Pp. 128, with 23 figures and 24 tables. 1959. Stockholm: Acta Chirurgica Scandinavica, Supplementum 242. Price Sw. kr. 20.

This is a report on an extensive piece of clinical research in Helsinki, in which arterial inadequacy in the lower limb was studied in patients the functional capacity of whose legs was tested on a bicycle ergometer. In order to test the circulatory deficit as thoroughly as possible the cycling was done with the legs elevated, pedalling upwards. All the usual methods of investigation were used, such as skin temperature estimations, in addition to studies of the colour of the soles and toes, and other obvious clinical testing. Normal persons were used as controls throughout the research. This work is described in great detail, and Lindström establishes his point that functional testing of this type is quite the most informative way of investigating chronic ischaemia in the lower limb. The responses of patients with various types of circulatory block are fully described. This is not an easy book to read, but its study is strongly recommended to all surgeons interested in circulatory disorders.—D. Ll. GRIFFITHS.

The Formation of Vein Thrombi Following Tissue Injury. An Experimental Study in Rabbits. By Stig BORGSTRÖM, Lars-Erik GELIN and Bengt ZEDERFELDT. $9\frac{1}{2} \times 7$ in. Pp. 36, with 14 figures. 1959. Stockholm: Acta Chirurgica Scandinavica, Supplementum 247. Price Sw. kr. 12.

This interesting and succinct monograph describes an investigation into the influence of tissue injury upon venous thrombosis. The authors used no less than 430 rabbits, of both sexes, which were allowed to move about freely. Venous stasis was produced by ligating both femoral veins and, in one series of animals, by ligating a large tributary as well. The muscles of one hind limb of each anaesthetised animal were then contused by light blows with a padded hammer, and the severity of the trauma was assessed by the number of blows. Not surprisingly, contusion was followed by an increasing tendency to thrombosis, and the number of thrombi increased with the severity of the injuries. The tendency to thrombosis lasted some weeks after the trauma, the duration again varying with the severity of the trauma. The effects of anaemia and of blood loss were studied, as were changes in the blood itself during the thrombotic phase and the mode of action of anticoagulants. This is a useful and timely study with obvious clinical significance, and is a piece of work of the high standard to be expected in a report in a supplement of *Acta Chirurgica Scandinavica*.—D. Ll. GRIFFITHS.

Bonding of Fractures by Plastic Adhesives, With Further Applications in a Research Project. By Bernard BLOCH, M.B., Ch.B.(Rand.), F.R.C.S.(Eng.). 10×7½ in. Pp. 23, with 24 figures. Sydney, Australia: Distributed by the Surgical Research Centre, Prince of Wales Division of Sydney Hospital.

This is a brief report of preliminary experiments with an industrial adhesive, an epoxy resin, which has been used in the repair of fractures of long bones in sheep and in man. An outline of the general chemistry of epoxy resins is given and the toxicity of certain constituents of the adhesive is mentioned. A full analysis of the particular resins employed is not given. The curing or polymerisation of an epoxy system is an exothermic reaction, but with the particular resins used the temperature rise does not seem to have been of importance. No toxic effects have been demonstrated in any of the experiments. The ingredients of the adhesive were supplied in collapsible tubes, the resins being mixed with the catalyst about eight minutes before use. The setting of the adhesive occurred twenty-three to twenty-five minutes later. The fracture site was exposed in the usual way and the bone cleared of all tissue, including the periosteum, for two inches on either side of the fracture. In the early experiments a polythene intramedullary plug was used. A thick adhesive mix was applied to the bone ends. Glass-fibre cloth impregnated with a thin resin mix was then applied around the bone. The adhesive was used in man in the treatment of twenty-three fractures of long bones. Some post-operative inflammatory reactions with bone resorption occurred. In seven cases the patients were able to resume full activity and there was both radiological and clinical evidence of bony union. In the other sixteen cases pseudo-arthrosis, infection, sequestration and mechanical failure of the glue occurred.

The experimental work so far reported does not justify at the present time the further use of this adhesive for the treatment of fractures in man.—John T. SCALES.

The Royal Edinburgh Hospital for Sick Children, 1860–1960. By Douglas GUTHRIE, M.D., F.R.C.S.Ed., F.R.S.E., Consulting Aural Surgeon, Royal Edinburgh Hospital for Sick Children. With seven other contributors. 9×6½ in. Pp. xii+75, with 34 figures. Index. 1960. Edinburgh and London: E. & S. Livingstone Ltd. Price 17s. 6d.

A centenary is a good excuse for looking back to the beginning and then trying to look into the future. Dr Douglas Guthrie has gathered together the facts about the foundation of the " Sick Kids " in Edinburgh with some short reminiscences, both of the staff and by the staff, administrative, medical and nursing. The change in the type of patient and the methods of treatment during what is historically so short a period as one hundred years, would be startling were it not commonplace. One hardly dares to imagine what change the next century will bring.

Professor R. W. B. Ellis points out one possible development for the immediate future when he writes of " improving child health throughout the world." Edinburgh has the duty to do in the backward areas of the world what its medical school, of which this hospital is a part, has done in the city's own back streets.

Orthopaedic surgeons may be interested to notice that except for the appointment of a plastic surgeon after World War II and of a chaplain and a psychiatrist following the establishment of the National Health Service, the orthopaedic department was the last recognised special department to be given its own specific head, an appointment made as recently as 1937. Nevertheless a most important place is occupied by two of the Hospital's surgeons in the story of bone and joint tuberculosis. Harold Stiles and his pupil and successor John Fraser gathered much of their experience at this Hospital, two out of the very distinguished staff which has given the Hospital its great reputation for teaching and for treatment.—A. White FRANKLIN.

Outline of Orthopaedics. By John Crawford ADAMS, M.D., F.R.C.S., Consultant Orthopaedic Surgeon, St Mary's Hospital, London, and St Vincent's Orthopaedic Hospital, Pinner; Assistant Editor, *Journal of Bone and Joint Surgery*. Third edition. 9×6 in. Pp. vii+440, with 313 figures. Index. 1960. Edinburgh and London: E. & S. Livingstone Ltd. Price 35s.

That *Outline of Orthopaedics* should have reached its third edition within four years of its birth is testimony enough to its popularity amongst undergraduate medical students, for whom it is primarily intended; the contents of this book are more than adequate for the qualifying examinations.

The present edition, which follows exactly the pattern of the last, has twelve pages more, because of the addition of a dozen illustrations, an increased size in some of the original illustrations and some addition to the text—notably a discussion of electro-diagnosis in neuromuscular conditions and a fuller

description of treatment by manipulation. That the book should not be enlarged greatly is not surprising, because in recent years there has been hardly anything new in the orthopaedic world for undergraduate consumption.

I enjoyed reading *Outline of Orthopaedics*; I found it well illustrated, concise and reasonably accurate, which one would expect from this clear-thinking author. The sections on clinical examination are really first class. There are a few minor criticisms one could offer: for example, the differential diagnosis of a lateral protrusion of a cervical disc is discussed but not that of a central one; and it was surprising to read that a tuberculous abscess should be drained.

It is to be hoped that in future editions this book will not expand, but that every endeavour will be made to keep it within reasonable bounds for the medical student.—David TREVOR.

Body Fluids in Surgery. By A. W. WILKINSON, Ch.M., F.R.C.S.E., F.R.C.S., Nuffield Professor of Paediatric Surgery, The Institute of Child Health of the University of London; Surgeon, The Hospital for Sick Children, Great Ormond Street, London. Second edition. 9×6 in. Pp. xi+276, with 12 figures and 30 tables. Index. 1960. Edinburgh and London: E. & S. Livingstone Ltd. Price 21s.

In the four years since its original publication Professor Wilkinson's monograph has become established as one of the outstanding British texts on problems of body fluid in surgery, and its virtues are widely recognised. In this new edition the whole text has been thoroughly revised to incorporate recent advances. In addition to including new material about the problems of surgery in hot climates, two major alterations have been made. First, the section on acid-base balance has been rewritten in terms of the Bronsted-Lowry convention, with a consequent emphasis on the H-ion. Professor Wilkinson is to be congratulated on this alteration. The basic concepts involved in the understanding of acid-base balance are not easy, but hitherto their comprehension has been made unduly difficult by the confusing terminology in which they were discussed, and this chapter should be of great assistance to anyone wishing to understand the processes involved in the maintenance of acid-base balance. Secondly, there is a new chapter on fluid balance in paediatric surgery. This chapter emphasises that in infants the problems differ not only quantitatively but also qualitatively from those in adults, and the careful detailed information will be of value to all those responsible for the surgical care of young children.

This monograph is an excellent, up-to-date, concise review of an increasingly important aspect of surgical care. Although in places the language is obscure, in general the information is clearly set out, and this book can be highly recommended as a guide to the physiology and pathology of the body fluids.—L. P. LE QUESNE.

Rose and Carless Manual of Surgery. Consulting Editor: Sir Cecil WAKELEY, *Bt., K.B.E., C.B.*, LL.D., D.Sc., M.Ch., F.R.C.S., F.R.S.E., F.A.C.S., F.R.A.C.S., Fellow of King's College, London; Past President of the Royal College of Surgeons of England; Consulting Surgeon, King's College Hospital, and to the Royal Navy. Editors: Michael HARMER, M.A., M.B.(Cantab.), F.R.C.S., Surgeon, Royal Marsden Hospital and Institute of Cancer Research (Royal Cancer Hospital), Paddington Green Children's Hospital and St Andrew's Hospital, Dollis Hill, London; and Selwyn TAYLOR, M.A., D.M., M.Ch.(Oxon.), F.R.C.S., Surgeon, King's College Hospital, Belgrave Hospital for Children, and Hammersmith Hospital; Lecturer in Surgery, Postgraduate Medical School, London. Assisted by fifteen contributors. Nineteenth edition. 10×7 in. Pp. xv+1,389, with many figures. 1960. London: Baillière, Tindall and Cox. Price 84s.

The nineteenth edition of Rose and Carless's Manual of Surgery has fifteen contributors, two editors and a consulting editor. The regional layout, form and illustrations of previous editions are recognisable, but the text has largely been rewritten and new illustrations added, with sections on electrolyte deficiencies, surgical infection and chemotherapy. Orthopaedics, including injuries, are well covered in 350 pages, about a quarter of the book, peripheral nerve injuries being included separately under the nervous system. This remains a sound general text-book of surgery particularly intended for final year students and postgraduates but also a good reference book for surgeons wishing to refer to progress in other fields of surgery. The text-book is now published in one volume instead of two, but remains of reasonable size, and I think this adds to its attraction.—Charles MANNING.

BOOKS RECEIVED

These books are acknowledged with grateful thanks. Selections will be made for review as space permits and in accordance with their interest for our readers.

Biophysical Investigations of the Mineral Phase in Healing Fractures. By Ulf NILSONNE. 1959. Copenhagen: Ejnar Munksgaard.

Consultations de Rhumatologie. By François FRANÇON. 1959. Paris: Editions Varia.

Orthopädische Technik. By Georg HOHMANN. 1958. Stuttgart: Ferdinand Enke Verlag.

Tabulating Equipment and Army Medical Statistics. By Albert G. LOVE, Eugene L. HAMILTON and Ida Levin HELLMAN. 1958. Washington: U.S. Government Printing Office.

Il Gargoilismo. By Guiseppe FALCHI and Mario MIDULLA. 1958. Rome: " Il Pensiero Scientifico " Editore.

Prosthetic Principles—Above Knee Amputations. By Miles H. ANDERSON, Ed.D., John J. BRAY, C.P., C.O., and Charles A. HENNESSY, C.P., C.O. Edited by Raymond E. SOLLARS. 1960. Springfield, Illinois: Charles C. Thomas, Publisher. Oxford: Blackwell Scientific Publications Ltd.

Osteochondritis Dissecans—Loose Bodies in Joints—Etiology, Pathology, Treatment. By I. S. SMILLIE, *O.B.E.*, Ch.M., F.R.C.S.(Ed.), F.R.F.P.S. 1960. Edinburgh and London: E. & S. Livingstone Ltd.

Nederlands Leerboek voor Heilgymnastiek en Massage. Onder redactie van Dr G. CHAPCHAL, P. H. van SETTEN arts, P. POLS en P. M. ZANDBOER. 1959. Amersfoort: Nederlands Genootschap voor Heilgymnastiek, Massage en Physiotechniek.

Surgery of Repair as Applied to Hand Injuries. By B. K. RANK, *C.M.G.*, M.S., F.R.C.S., F.R.A.C.S., and A. R. WAKEFIELD, M.S., F.R.C.S., F.R.A.C.S. With a foreword by Sir Gordon GORDON-TAYLOR, *K.B.E.*, *C.B.*, F.R.C.S. Second edition. 1960. Edinburgh and London: E. & S. Livingstone Ltd.

The Foot and Ankle. Their Injuries, Diseases, Deformities and Disabilities. By Philip LEWIN, M.D., F.A.C.S., F.I.C.S. Fourth edition. 1959. London: Henry Kimpton.

Spinal Joint Changes and Dental Infections. By Bo E. INGELMARK, Vilh. MØLLER-CHRISTENSEN and Ove BRINCH. 1959. Basel/New York: S. Karger.

Active Alerted Posture. By W. E. TUCKER. 1960. Edinburgh and London: E. & S. Livingstone Ltd.

Lehrbuch der Orthopädie und Traumatologie. By Max LANGE. Band I: Allgemeine Orthopädie/Angeborene Erkrankungen und Leiden. 1960. Stuttgart: Ferdinand Enke Verlag.

A Clinical Prospect of the Cancer Problem. By D. W. SMITHERS, M.D., F.R.C.P., F.F.R. Introductory Volume of Monographs on Neoplastic Disease at Various Sites. 1960. Edinburgh and London: E. & S. Livingstone Ltd.

Bone as a Tissue. Edited by Kaare RODAHL, M.D., Jesse T. NICHOLSON, M.D., and Ernest M. BROWN, Jun., M.D. 1960. New York, Toronto and London: The Blakiston Division McGraw-Hill Book Company, Inc.

Prophylaxie et Traitement des Escarres de Décubitus. By R. VILAIN. 1960. Paris: Expansion Scientifique Française.

Fractures and Orthopaedic Surgery for Nurses and Physiotherapists. By Arthur NAYLOR, Ch.M., M.B., M.Sc., F.R.C.S. With a foreword by Sir Ernest FINCH, M.D., M.S., F.R.C.S. Fourth edition. 1960. Edinburgh and London: E. & S. Livingstone Ltd.

Structure and Function of Muscle. Edited by G. H. BOURNE. Volume I: Structure. 1960. New York and London: Academic Press Inc., Publishers.

Surgery in World War II. Neurosurgery—Volume II. Edited by R. Glen SPURLING, M.D., and Barnes WOODHALL, M.D. 1959. Washington: U.S. Government Printing Office.

French's Index of Differential Diagnosis. Edited by Arthur H. DOUTHWAITE, M.D., F.R.C.P. Eighth edition. 1960. Bristol: John Wright & Sons Ltd.

Elongation Treatment of Low Back Pain. By George H. HASSARD, M.D., and Charles L. REDD, B.S., P.T. With forewords by Harvey E. BILLIG, Jun., M.D., F.I.C.S., F.A.C.S.M., and R. S. MINSKER. 1960. Springfield, Illinois: Charles C. Thomas, Publisher. Oxford: Blackwell Scientific Publications Ltd.

Ergebnisse der Wirbelsäulenforschung. Zusammengestellt von Prof. Dr med. H. JUNGHANNS. 1960. Stuttgart: Hippokrates Verlag.

The Pathology of Tumours of the Nervous System. By Dorothy S. RUSSELL, Sc.D., M.A., M.D., F.R.C.P., LL.D., and L. J. RUBINSTEIN, M.D. With a chapter on Tissue Culture in Relation to Tumours of the Nervous System by C. E. LUMSDEN, M.D. 1959. London: Edward Arnold (Publishers) Ltd.

Surgery in the Aged. Edited by Frank GLENN, M.D., S. W. MOORE, M.D., and John M. BEAL, M.D. 1960. New York, Toronto and London: McGraw-Hill Book Company Inc.

A Catalogue of the H. Winnett Orr Historical Collection and Other Rare Books in the Library of the American College of Surgeons. Edited by L. Margueriete PRIME. 1960. Chicago: American College of Surgeons.

ACKNOWLEDGMENTS

We acknowledge with thanks receipt of:

Acta Chirurgica Scandinavica, and Supplements
Acta Orthopaedica Italica
Acta Ortopedica-Traumatologica Iberica
Annals of the Royal College of Surgeons of England
Archives of Surgery
Biological Abstracts
Boletin de la Sociedad de Cirugia del Uruguay
British Journal of Surgery
British Medical Bulletin
Canadian Journal of Surgery
Indian Journal of Surgery
Journal of Neurology, Neurosurgery and Psychiatry
Journal of the American Medical Association
Journal of the Royal College of Surgeons of Edinburgh
Lancet
Ortopedia e Traumatologia dell'Apparato Motore
Radiology
Revista de Ortopedia y Traumatologia
Revue de Chirurgie Orthopédique
Surgery, Gynecology and Obstetrics
Zeitschrift für Orthopädie und ihre Grenzgebiete

AUTHOR INDEX

A

Alldred, A. Localised ischaemia of calf, 659.
Alms, M. Medullary nailing, 398.
Amato, V. P. *See* Trueta, J., and Amato, V. P.
Andrews, P. S. *See* Wiles, P., Andrews, P. S., and Bremner, R. A.
Angliss, E. Extreme hypertrophic bone reaction after medullary nailing, 409.
Arden, G. P. Radioactive isotopes in fractures of neck of femur, *21.
Value of radioactive isotopes in determining viability of bone (*discussion*), 650.
Aston, J. N. Locked middle finger, *75.

B

Bär, G. F. J. M. Prolapsed intervertebral disc in the adolescent (*discussion*), 651.
Shelf operation in osteoarthritis of hip, 653.
Bain, A. Movements of big toe in flat feet, 159.
Ball, J. Morbid anatomy of renal osteodystrophy, 158.
Barnes, R. Alexander Miller (*obituary*), 150.
Book review, 872.
Injuries of sciatic nerve (*editorial*), 175.
Barry, H. C. *Discussions*, 404, 406, 407, 409.
Transthoracic spinal fusion, 406.
Bauer, G. C. H., and Wendeberg, B. Bone-seeking isotopes as indicators of bone turnover, 397.
Beasley, A. W. Non-tuberculous infection of spine, 660.
Bedbrook, G. M. *Discussions*, 402, 404.
Multiple congenital deformities, 405.
and Doyle, W. A. Aid for severely paralysed upper extremities, *358.
Beddow, F. H., and Corkery, P. H. Lateral dislocation of radio-humeral joint with greenstick fracture of upper end of ulna, *782.
Bensted, J. P. M., and Blackett, N. M. Bone tumour production: dose response relationships using ^{32}P, 394.
Bényi, P. Modified Lambrinudi operation for drop foot, *333.
Bertelsen, A. Sympathectomy for diseases of extremities, 661.
and Capener, N. Fingers, compensation and King Canute, *390.
Betts, W. Unreduced dislocation of radius in Monteggia fracture-dislocation (*discussion*), 406.
Biggart, J. H. *Book review*, 415.
Blackett, N. M. *See* Bensted, J. P. M., and Blackett, N. M.
Blackwood, W. *Book review*, 873.
Bloch, B. Fixation of fractures by plastic adhesives, 407.
Blockey, N. J., and Schorstein, J. Stiff back in children, 651.
Blunden, R., and Gibson, R. Anteversion of neck of femur, 659.
Bobechko, W. P., and Harris, W. R. Radiographic density of avascular bone, *626.
Bohr, H. H. Microradiography in patients with bone disease, 663.
Bolton, H. Intraneural ganglion of lateral popliteal nerve (*discussion*), 652.
Primary treatment of tendon injuries, 647.
Bonney, G. L. W. *Book review*, 415.
Use of intravenous dye technique in assessment of tissue viability (*discussion*), 646.
Bonnin, J. G. *Book reviews*, 168, 666.
Bonnin, L. *Discussions*, 403, 410.
Bose, K. S. Atypical osteomyelitis simulating bone tumours and other diseases, 407.
Bremner, R. A. *See* Wiles, P., Andrews, P. S., and Bremner, R. A.
Brett, M. S. Nature of remodelling after femoral supracondylar osteotomy in childhood, 653.
Brewerton, D. A. *Book reviews*, 664, 665.
Brock, B. H. Report of 1959 Travelling Scholar, 655.
Brodetti, A. Blood supply of femoral neck and head in relation to damaging effects of nails and screws, *794.
Brook, A. L. Eyre-. *See* Eyre-Brook, A. L.
Brookes, M. Vascular reaction of tubular bone to ischaemia in peripheral occlusive vascular disease, *110.
Brooks, D. M. *Book review*, 664.
Symposium on hand surgery: value of nerve repair, 647.
Brown, M. Forrester-. *See* Forrester-Brown, M.
Bruser, D. M. Direct lateral approach to lateral compartment of knee joint, *348.
Bryant, F. J., and Peirson, D. H. ^{90}Strontium in the environment and human bone, 395.
Buck, J. E. *Discussions*, 158.
Burger, M., and Sobel, A. E. Calcification *in vitro* and phosphatase activity, *137.
Burrows, H. J. *Book review*, 667.
Bywaters, E. G. L. *Book review*, 165.

C

Callow, C. *Discussions*, 407, 408.
Campbell Golding, F. *See* Golding, F. C.
Capener, N. Avascular necrosis and the head of the femur (*discussion*), 650.
Book reviews, 174, 413, 417, 421, 666.
Symposium on hand surgery, 649.
See also Bertelsen, A., and Capener, N.
Carter, C., and Sweetnam, D. R. Recurrent dislocation of patella and of shoulder: association with familial joint laxity, *721.
See also Wilkinson, J. A., and Carter, C.
Catto, M. E. *Book review*, 170.
Histopathology of renal osteodystrophy, 157.
Chalmers, J. *Book review*, 872.
Growth of transplanted foetal bone, 653.
and Coulson, W. F. A metastasising chordoma, *556.
Charnley, J. Anchorage of femoral head prosthesis to shaft of femur, *28.
Book reviews, 168, 865.
Surgical treatment of ununited fractures (*editorial*), 3.
Symposium on hand surgery: replacement of lost skin (*discussion*), 646.
Claffey, T. J. Avascular necrosis of femoral head, *802.
Open fractures of tibia, 407.
Clarke, H. Osmond-. *See* Osmond-Clarke, H.
Clawson, D. K., and Seddon, H. J. Late consequences of sciatic nerve injury, *213.
Results of repair of sciatic nerve, *205.

* Indicates full article.

Clennar, G. *See* Hosking, G. E., and Clennar, G.
Cloke, J. *Discussions*, 403, 406.
Cockshott, W. P., and Davies, A. G. M. Tumoural gummatous yaws, *785.
Cohen, B. Keon-. *See* Keon-Cohen, B.
Cohen, H., 1st Baron of Birkenhead. Reflections on specialism in medicine (*Watson-Jones Lecture*), 153.
Coleman, H. M. Injuries of articular disc at wrist, *522.
Collins, D. H. *Book review*, 171.
Colonna, P. C. Trochanteric reconstruction operation for ununited fractures of upper end of femur, *5.
Colquhoun, J. B. *Australian Orthopaedic Association, presidential address*, 400.
Bilateral osteochondritis dissecans, 410.
Discussions, 404, 405.
Coltart, W. D. *Book reviews*, 174, 864.
Corkery, P. H. *See* Beddow, F. H., and Corkery, P. H.
Coulson, W. F. *See* Chalmers, J., and Coulson, W. F.
Courts, A. *See* Kelly, M. E., and Courts, A.
Critchley, J. Discitis, 410.
Cromack, A. Chronic bone abscess, 404.
Cruickshank, B. *See* Murray, A. M., and Cruickshank, B.

D

Darling, A. I. Organic matrix of dental enamel in some diseases, 854.
Davies, A. G. M. *See* Cockshott, W. P., and Davies, A. G. M.
Dawkins, A. (*Adelaide*). Talipes equinovarus, 407.
Dawkins, A. (*Perth*). Morton's metatarsalgia (*discussion*), 403.
Tumours of head of humerus, 405.
Dawson, J. Effect of parathormone (Lilley) on oxidative phosphorylation, 155.
Dawson, R. H. Review of fifty Judet arthroplasties, 660.
Daymond, K. *Demonstrations*, 406, 411.
Discussions, 402, 404.
De Souza, W. *Demonstrations*, 400.
Degenhardt, D. P., and Goodwin, M. A. Neuropathic joints in diabetes, *769.
Desbrosses, J. *See* Stagnari, P., *et al.*
Devas, M. B. Longitudinal stress fractures, *508.
Stress fractures of patella, *71.
Dickerson, J. W. T., and Widdowson, E. M. Accelerated growth and skeletal development in the rat, 156.
Dickson, J. W. *See* Scott, J. H. S., *et al.*
Diveley, R. L. Cervical disc syndrome (*discussion*), 860.
Diagnosis and treatment of the painful back, 859.
Fracture of scaphoid bone, 860.
Doig, W. G. *Demonstrations*, 410.
See also Scott, J. H. S., *et al.*
Dommisse, G. F. Diametric fractures of pelvis, *432.
Dooley, B. *Demonstrations*, 410.
Irreducible postero-lateral dislocation of knee, 409.
Doyle, W. A. *See* Bedbrook, G. M., and Doyle, W. A.
Du Toit, G. T. Multiple epiphysial dysplasia (*discussion*), 863.
Reduction of frequency and severity of injury in car accidents, 862.
and Roux, D. Operative treatment of prolapsed lumbar discs, 860.
Dunoyer, J. *See* Stagnari, P., *et al.*
Durbin, F. C. Treatment of slipped upper femoral epiphysis, *289.
Dwyer, A. *Discussions*, 401, 403, 404, 405.
Dwyer, F. Treatment of relapsed club foot, 858.

E

Enklaar, J. E. Legg-Perthes' disease: etiology and treatment, 651.
Epstein, B. Recent advances in cerebral palsy, 861.
Eriksen, B. Lumbo-sacral fusion, 660.
Esteve, R. Congenital dislocation of hip, *253.
Evans, D. Eosinophilic granuloma of bone, 399.
Evans, R. W., Thomas, G. E., and Walker, N. M. Congenital malignant synovial tumour of bone, *742.
Eyre-Brook, A. L. *Book review*, 414.
Intraneural ganglion of lateral popliteal nerve (*discussion*), 652.
Septic arthritis of hip and osteomyelitis of upper end of femur in infants, *11.
Synovial sarcoma (*discussion*), 650.

F

Fabry, C. Chemical composition and histological structure of bone, 856.
Fairbank, T. J. *Book reviews*, 420, 870.
Farrington, G. D. Léri's disease, 857.
Fish, H. *Book review*, 667.
Fisher, J. H. *See* Kennedy, J. C., and Fisher, J. H.
Flint, M., and Sweetnam, D. R. Amputation of all toes, *90; (*correspondence*), 412.
Forrester-Brown, M. Treatment of calcaneus foot (*discussion*), 862.
Fowler, A. W. Abdominal neuromyotomy, 398.
Amputation of all toes (*correspondence*), 412.
Forefoot reconstruction, 399.
Frankel, M. *See* Weinberg, H., *et al.*
Frankel, V. H. *See* Hirsch, C., and Frankel, V. H.
Franklin, A. W. *Book review*, 875.
Fraser, R., and Harrison, M. Clinical diagnosis of renal osteodystrophy, 156.
Furlong, R. *Book review*, 865.

G

Gayton, J. *Demonstrations*, 409.
Gayton, W. *Demonstrations*, 406.
Gervis, W. H. *Demonstrations*, 158.
Gibson, J. M. C. Multiple injuries: management of the patient with a fractured femur and a head injury, *425.
Gibson R. *See* Blunden, R., and Gibson, R.
Giles, K. W. Anatomical variations affecting surgery of de Quervain's disease, *352.
Gillespie, H. W. *Book review*, 416.
Gilmour, W. Congenital pseudarthrosis of tibia, 404.
Discussions, 402, 404.
Golding, F. C. Museum pages:
I—Changes in knees in ankylosing spondylitis or Reiter's syndrome, *142.
II—Cysts of lateral semilunar cartilage of knee, *144.
III—Spina bifida and epiphysial displacement, *387.
IV—Differential diagnosis of Paget's disease, *641.
V—Sesamoids of hallux, *840.
Goodwin, M. A. *See* Degenhardt, D. P., and Goodwin, M. A.
Griffiths, D. Ll. *Book reviews*, 163, 165, 420, 874.
Gruca, A. Structural scoliosis, 160.

H

Hall, M. C., and Pennal, G. F. Primary subtalar arthrodesis in treatment of severe fractures of calcaneum, *336.

* Indicates full article.

Hall, R. McK. Chronic septic arthritis caused by foreign bodies (*discussion*), 405.
Renal rickets, 404.
Hambury, H. J. Recurrent dislocation of elbow, 399.
Hamilton, A. *Discussions*, 401, 403, 404, 405, 410.
Osteochondritis dissecans of both knees, 405.
Hardy, A. Treatment of paraplegia from fracture-dislocation of thoraco-lumbar spine, 661.
Harris, N. H. Some problems in diagnosis and treatment of acute osteomyelitis, *535.
Harris, W. H. Microscopic method of determining rates of bone growth, 856.
Harris, W. R. *See* Bobechko, W. P., and Harris, W. R.
Harrison, G. E. Retention of an intravenous dose of radioactive strontium in man, 395.
Harrison, M. *See* Fraser, R., and Harrison, M.
Harrison, R. G. *Book review*, 419.
Pearson, M. A., and Roaf, R. Ulnar dimelia, *549.
Harrold, A. J. Failure of union in fracture of neck of femur, *226.
Hedden, F. J. Treatment of calcaneus foot, 861.
Helfet, A. J. Fracture of scaphoid bone (*discussion*), 861.
Hendry, N. G. C., and Mitchell, P. E. G. Chemistry of intervertebral disc prolapse, 855.
Henry, W. L. Crushed chest: management of flail anterior fragment (*discussion*), 652.
Hill, H. Osteochondritis dissecans of both knees (*discussion*), 406.
Pseudarthrosis of neck of femur, 403.
Hill, R. C. J. Cervical disc syndrome, 860.
Fracture of scaphoid bone (*discussion*), 861.
Hirsch, C., and Frankel, V. H. Analysis of forces producing fractures of proximal end of femur, *633.
Hodgkinson, A. Low posterior approach to hip joint, 408.
Hodgkinson, R. *Discussions*, 404, 405, 406, 409.
Hoets, J. John Rudolph Sergius Lahz (*obituary*), 644.
Hogg, T. Osteoarthritis of knee after meniscectomy (*discussion*), 408.
Holdsworth, F. W. *Book reviews*, 170, 866.
Holgate, W. Preliminary report on uptake and retention of ^{90}Sr in teeth and jaws of rabbits, 156.
Holmes, J. R. Avian osteopetrosis, 855.
Hooper, R. Spinothalamic tractotomy for relief of pain in osteoarthritis of hip, 409.
Hosking, G. E., and Clennar, G. Calcification in articular cartilage, *530.
Howard, R. Haemangioma, 857.
Hudson, C. Fatigue fractures, 405.
Hueston, J. Biceps cineplasty (*film*), 407.
Husain, F. Chordoma of thoracic spine, *560.
Hutchison, J. Extractor for Moore's pins, *570.
and Park, W. W. Chondromyxoid fibroma of bone, *542.

I

Innes, A. P. Rose-. *See* Rose-Innes, A. P.

J

Jackson Burrows, H. *See* Burrows, H. J.
Jacobsen, A. Vitallium cup arthroplasty in osteoarthritis of hip, 403.
James, J. I. P. *Book review*, 871.
Late functional and economic results of major hand injuries (*discussion*), 648.
James, N. E. *Demonstrations*, 858.
Jarry, L., and Uhthoff, H. K. Activation of osteogenesis by the " petal " technique, *126.
Jayes, P. H. *Book review*, 665.
Jebens, E. H. *Book review*, 172.
Jeffery, C. C. Place of open operation in treatment of fractures of hand, 647.
Jens, J. Resection of tumours (*discussion*), 406.
Jensen, E. *See* Mortens, J., and Jensen, E.
Jones, D. M. Internal fixation for McMurray osteotomy, 398.
Jones, G. B. *Book review*, 418.
Jones, R. Conservative treatment of congenital dislocation of hip, 401.
Jowsey, J. Variations in structure of normal bone, 856.

K

Kallio, K. *Discussions*, 401, 403, 404, 405, 406.
Use of free skin, 403.
Keir, D. M. Medullary nailing for delayed healing of fractures of femur, 658.
Kellock, W. Thompson's prosthesis, 857.
Kelly, J. Congenital dislocation of hip, 858.
Kelly, M. E., and Courts, A. Changes in bone matrix, 855.
Kember, N. F. Cell division in endochondral ossification, *824.
Use of tritiated thymidine in study of bone, 857.
Kennedy, J. C., and Fisher, J. H. Haemangiopericytoma, *80.
Kent, P. W. Chemical constituents of bone matrix, 853.
Keon-Cohen, B. *Demonstrations*, 409.
Kerridge, G. Instability of fifth cervical vertebra on sixth after car accident, 405.
Morton's metatarsalgia, 403.
Ununited clavicle (*discussion*), 406.
Kessel, L. Sublimis tendon replacement technique in tendon injuries (*discussion*), 647.
King, A. B. Tumours of forearm, 400.
King, T. Chondroblastoma—chondrosarcoma, 409.
Discussions, 403, 404, 406.
Kingma, M. S. Hetero-transplants and homo-transplants of bone, 653.

L

Lack, C. H. *Book review*, 420.
Lacroix, P. Anatomical sites of deposition of ^{45}Ca in bone, 396.
Lahz, J. *Discussions*, 402, 406.
Lamerton, L. F. Biological implications of radiation dosage to bone from environmental radioactive material, 396.
Lancaster, J. M. Prolapsed intervertebral disc in the adolescent (*discussion*), 651.
Lancaster, R. Percy-. *See* Percy-Lancaster, R.
Landells, J. W. Histological abnormalities in matrix of bone and cartilage, 854.
Le Quesne, L. P. *Book review*, 876.
Lewin, W. *Book review*, 167.
Lipmann Kessel. *See* Kessel, L.
Little, K. Fine structure of bone, cartilage and tooth matrix, 853.
See also Trueta, J., and Little, K.
Lloyd-Roberts, G. C. Suppurative arthritis of infancy, *706.
London, P. S. Simplicity of approach in treatment of hand injuries, 648.
Lumb, F. H. Early manifestations of renal osteodystrophy, 157.

* Indicates full article.

M

McCormack, M. P. Upper lumbar chordoma, *565.
Macdonald, W. L. Osteoarthritis of knee after meniscectomy (*discussion*), 409.
McDougall, A., and Robertson, J. M. Prolapsed intervertebral disc in the adolescent, 651.
McFarland, W. J. B. Central displacement stabilising operation on hip, 659.
McGann, T. C. A. Influence of citrate on composition and form of calcium phosphate precipitates, 855.
McGregor, I. A. Symposium on hand surgery: replacement of lost skin, 646.
McKee, G. K. Artificial joints, 857.
McKellar, C. Bone comminution from saw cut, 406.
Congenital pseudarthrosis of tibia (*discussion*), 404.
MacKenzie, I. G. Arthrodesis of wrist in reconstructive surgery, *60.
Seddon, H. J., and Trevor, D. Congenital dislocation of hip, *689.
Macnab, I. *Book review*, 419.
McNeur, C. *Discussions*, 402, 404.
Macpherson, S., Owen, M., and Vaughan, Dame J. Relation of radiation damage to radiation dose from ^{90}Strontium, 395.
McSweeney, A. *Discussions*, 403, 405, 406, 409, 410.
Medial and posterior release operation for club foot, 402.
Makin, M. *See* Weinberg, H., *et al.*
Manning, C. W. S. F. *Book reviews*, 869, 876.
Martin, D. J. *Demonstrations*, 159.
Maxwell, C. M. Orthopaedic aspects of resistant rickets, 409.
Mayer, J. H. Birth palsy (*discussion*), 158.
Menelaus, M. B. *See* Simmonds, F. A., and Menelaus, M. B.
Mercer, Sir W. *Book review*, 163.
Some Edinburgh pioneers in orthopaedic surgery, 393.
Merryweather, R. Synovial cysts of fingers, 399.
Miller, J. E. Javelin thrower's elbow, *788.
Mitchell, P. E. G. *See* Hendry, N. G. C., and Mitchell, P. E. G.
Mol Van Oterloo, J. de. Case for diagnosis, 652.
Moll, P. J. Synovial sarcoma, 650.
Moller, C. T. Treatment of calcaneus foot (*discussion*), 862.
Montgomery, R. D., and Standard, K. L. Albers-Schönberg's disease, *303.
Morgan, J. D., and Somerville, E. W. Normal and abnormal growth at upper end of femur, *264.
See also Trueta, J., and Morgan, J. D.
Morgan, N. H. Osteoarthritis of knee after meniscectomy (*discussion*), 408.
Morrison, A. Hyperextension injury of cervical spine with rupture of oesophagus, *356.
Mortens, J., and Jensen, E. Charnley stabilisation of hip, *466.
Mourgues, G. de. *See* Trojan, E., and Mourgues, G. de.
Muir, I. F. K. Late functional and economic results of major hand injuries, 648.
Murley, A. H. G. Excision of trapezium in osteoarthritis of first carpo-metacarpal joint, *502.
Murley, J. Tendon transfer for poliomyelitic paralyses, 410.
Murray, A. M., and Cruickshank, B. Dyschondroplasia, *344.
Murray, R. O. *Book reviews*, 171, 871.

N

Nelson, D. H. Forced inversion of ankles, *793.
Newman, P. H. *Book reviews*, 173, 873.
Surgical treatment of slipping of upper femoral epiphysis, *280.
Nicoll, E. A. Alexander Miller (*obituary*), 150.
Book review, 867.
Cancellous bone grafting in treatment of non-union of fractures of long bones, 662.
Treatment of fractures of thoraco-lumbar spine, 662.
Nisbet, N. W. Anatomy of calcaneal tendon of rabbit, *360.
Research in Department of Surgery of Medical School of University of Otago on basic problems in bone grafts, 659.
Rupture of tendo calcaneus, 659.
Nissen, K. I. Arrest of primary osteoarthritis of hip (*editorial*), 423.

O

O'Connell, J. E. A. *Book review*, 166.
O'Driscoll, R. F. Rural orthopaedic problems, 858.
Osborne, G. Intraneural ganglion of lateral popliteal nerve (*discussion*), 652.
Sublimis tendon replacement technique in tendon injuries, 647.
Osmond-Clarke, H. *Discussions*, 407, 409, 410.
Present position of surgery of hip and spinal column surgery, 659.
Sir Archibald Hector McIndoe (*obituary*), 844.
Oterloo, J. de Mol Van. *See* Mol Van Oterloo, J. de.
Owen, M. *See* Macpherson, S., Owen, M., and Vaughan, Dame J.

P

Pannell, J. Osteoarthritis of knee after meniscectomy, 408.
Park, W. W. *See* Hutchison, J., and Park, W. W.
Parker, D. *Discussions*, 407, 410.
Parkes, A. R. Intraneural ganglion of lateral popliteal nerve, 652.
Parnell, W. Brachial plexus injuries with vascular damage, 404.
Parry, C. B. Wynn. *See* Wynn Parry, C. B.
Parsons, J. Bone grafting, 862.
Flexion deformities of hip, 863.
Patrick, J. Avascular necrosis and the head of the femur, 650.
Pautard, F. G. E. Initiation and development of the inorganic phase of hard tissues, 155.
Pearson, M. A. *See* Harrison, R. G., Pearson, M. A., and Roaf, R.
Pedersen, E. K. Dysplasia epiphysialis capitis femoris, 663.
Peirson, D. H. *See* Bryant, F. J., and Peirson, D. H.
Pennal, G. F. *See* Hall, M. C., and Pennal, G. F.
Percy-Lancaster, R. Bilateral slipped upper femoral epiphyses, 863.
Bone grafting (*discussion*), 863.
Problems of tuberculosis in the Bantu as seen by an orthopaedic surgeon, 861.
Recurrent posterior dislocation of shoulder, 863.
Piggot, J. Charnley stabilisation of hip, *476.
See also Robins, R. H. C., and Piggot, J.
Piggott, H. Natural history of hallux valgus in adolescence and early adult life, *749.
Pike, W. *Demonstrations*, 410.
Platt, Sir H., Bart., *Book review*, 665.
Pioneers of rehabilitation (*Dame Georgiana Buller memorial lecture*), 852.
Plewes, L. W. *Book reviews*, 168, 169.
Powell, H. D. W. Simultaneous bilateral fractures of neck of femur, *236.
Price, C. H. G. Seasonal rhythm in presentation of human bone sarcoma, 156.

* Indicates full article.

Price, E. E. *Demonstrations*, 410.
Discussions, 403, 404, 406, 409.
Place of surgery in treatment of congenital dislocation of hip, 400.
Stiff back in children (*discussion*), 651.
Pritchard, A. E., and Thompson, W. A. L. Acute pyogenic infections of spine in children, *86.
Pulvertaft, R. G. Late functional and economic results of major hand injuries (*discussion*), 649.
Symposium on hand surgery: general review, 646.

Q

Queneau, P. *See* Stagnari, P., *et al.*

R

Raffler, W. Dislocation of hip, 857.
Ratliff, A. H. C. *See* Scott, J. H. S., *et al.*
Regan, B. F. Intertrochanteric osteotomy, 858.
Reid, D. A. C. Reconstruction of mutilated hand, 648.
Reconstruction of thumb, *444.
Reimann, I. Calvé-Perthes' disease, 661.
Rennie, A. M. Pathology of slipped upper femoral epiphysis, *273.
Rhydderch, A. I. Chronic septic arthritis caused by foreign bodies, 405.
Discussions, 402, 407.
Richards, H. J. Symposium on hand surgery: replacement of lost skin (*discussion*), 646.
Transthoracic surgery of spine, 399.
Roaf, R. *Book review*, 871.
Mechanics of spinal injuries, *810.
Vertebral growth and its mechanical control, *40.
See also Harrison, R. G., Pearson, M. A., and Roaf, R.
Roarty, J. Myelomatosis, 407.
Roberts, G. C. Lloyd-. *See* Lloyd-Roberts, G. C.
Robertson, J. M. *See* McDougall, A., and Robertson, J. M.
Robins, R. H. C., and Piggot, J. McMurray osteotomy, *480.
See also Scott, J. H. S., *et al.*
Rose-Innes, A. P. Anterior dislocation of ulna at inferior radio-ulnar joint, *515.
Roux, D. *See* Du Toit, G. T., and Roux, D.
Rushton, M. A. Radiation damage to dental tissues in high-dose rabbit experiments, 395.
Russell, R. S. Entry of ^{90}Strontium into human diet, 396.

S

Scales, J. T. *Book review*, 875.
Schorstein, J. *See* Blockey, N. J., and Schorstein, J.
Scott, J. C. Late functional and economic results of major hand injuries (*discussion*), 649.
Scott, J. H. S., *et al.* Visit of 1960 Travelling Fellows to United States and Canada, 847.
Scott Russell, R. *See* Russell, R. S.
Seddon, H. J. *See* Clawson, D. K., and Seddon, H. J.
See also MacKenzie, I. G., Seddon, H. J., and Trevor, D.
Shaw, N. E. Neonatal sciatic palsy from injection into umbilical cord, *736.
Sheard, E. *Demonstrations*, 863.
Shephard, E. *Demonstrations*, 158.
Simultaneous posterior dislocation of both shoulders, *728.
and Solomon, D. J. Carpo-metacarpal dislocation, *772.
Shepherd, M. M. Further review of results of operations on hip joint, *177.
Shiers, L. G. P. Arthroplasty of knee, *31.
Sillar, W. Crushed chest: management of flail anterior fragment, 652.
Simmonds, F. A., and Menelaus, M. B. Hallux valgus in adolescents, *761.
Simpson, B. S. Disappearing bones (*correspondence*), 162.
Smith, J. W. Arrangement of collagen fibres in human secondary osteones, *588.
Smith, N. *Demonstrations*, 399.
"Tennis" elbow, 400.
Tumours of forearm (*discussion*), 400.
Smyth, E. A. Amputation of legs, 159.
Sobel, A. E. *See* Burger, M., and Sobel, A. E.
Solomon, D. J. *See* Shephard, E., and Solomon, D. J.
Somerville, E. W. Congenital dislocation of hip (*discussion*), 652.
Flexion contractures of knee, *730.
See also Morgan, J. D., and Somerville, E. W.
Souza, W. de. *See* De Souza, W.
Spiers, F. W. Radiation dosage to bone from all sources, 397.
Spring, E. *Demonstrations*, 409, 410.
Stack, H. G. Place of open operation in treatment of fractures of hand (*discussion*), 647.
Stack, M. V. Changes in organic matrix of enamel during growth, 853.
Stagnari, P., *et al.* Structural scoliosis, 160.
Stanbury, S. W. Metabolic implications of therapy in azotaemic osteodystrophy, 157.
Standard, K. L. *See* Montgomery, R. D., and Standard, K. L.
Stanger, J. K. Charles Gordon Irwin (*obituary*), 148.
Stening, W. Treatment of congenital dislocation of hip (*discussion*), 401.
Stephenson, R. Dislocation of wrist, 400.
Stevens, J. Value of radioactive isotopes in determining viability of bone, 650.
Stirling, R. I. The mutability of living bone (*Lady Jones Lecture*), 153.
Storey, E. Osteosclerosis after intermittent administration of large doses of vitamin D in the rat, *606.
Strange, F. G. St C. Birth palsy (*discussion*), 158.
Swaney, W. Rotator cuff tears, 409.
Sweetnam, D. R. *See* Carter, C., and Sweetnam, D. R.
See also Flint, M., and Sweetnam, D. R.

T

Taylor, J. G. Congenital absence of radius, 857.
Congenital coxa vara, 857.
Tempest, M. N. Use of intravenous dye technique in assessment of tissue viability, 646.
Tennant, D. C. Colles's fractures, 159.
Thomas, G. E. *See* Evans, R. W., Thomas, G. E., and Walker, N. M.
Thompson, S. M. *Demonstrations*, 858.
Thompson, W. A. L. *See* Pritchard, A. E., and Thompson, W. A. L.
Toit, G. T. du. *See* Du Toit, G. T.
Trevor, D. *Book reviews*, 173, 875.
See also MacKenzie, I. G., Seddon, H. J., and Trevor, D.
Trojan, E., and Mourgues, G. de. Fractures of carpal scaphoid bone, 160.
Trueta, J. *Book review*, 413.
Disintegration of joint cartilage, 854.
and Amato, V. P. Vascular contribution to osteogenesis. III—Changes in growth cartilage caused by experimentally induced ischaemia, *571.
and Little, K. Vascular contribution to osteogenesis. II—Studies with electron microscope, *367.
and Morgan, J. D. Vascular contribution to osteogenesis. I—Studies by injection method, *97.

* Indicates full article.

Tubiana, R. Symposium on hand surgery: the neurovascular island flap, 647.
Tupman, G. S. An evaluation of bone stimulation for leg discrepancy, 398.
Treatment of inequality of lower limbs, *489.

U

Uhthoff, H. K. *See* Jarry, L., and Uhthoff, H. K.

V

Vas, E. *See* Weinberg, H., *et al.*
Vaughan, Dame J. *See* Macpherson, S., Owen, M., and Vaughan, Dame J.
Vaughan-Jackson, O. J. *Discussions*, 158.
Verbeek, O. Treatment of pseudarthroses and fractures of long bones by homogenous bone grafts, 653.
Verdan, C. E. Primary treatment of tendon injuries (*discussion*), 647.
Vieyra, H. J. K. *Demonstrations*, 658.
Vogel, J. Bilateral congenital dislocation of hips, 862.
Multiple epiphysial dysplasia, 862.

W

Wainwright, D. Shelf operation in osteoarthritis of hip (*discussion*), 653.
Wakefield, A. Tendon and nerve injuries at wrist and in palm, 408.
Walker, N. M. *See* Evans, R. W., Thomas, G. E., and Walker, N. M.
Walmsley, R. *See* Whiston, T. B., and Walmsley, R.
Watts, A. *Discussions*, 401, 403, 405.
Wayne, E. J. A new approach to diagnosis and treatment, 649.
Webb, A. L. *Discussions*, 407, 409, 410.
Weidmann, S. M. Studies on calcification of cartilage, 154.
Weinberg, H., *et al.* Familial epiphysial dysplasia of lower limbs, *313.
Wells, C. Neurological disorders that may appear in a foot clinic, 399.
Wendeberg, B. *See* Bauer, G. C. H., and Wendeberg, B.
West, E. F. Atypical osteomyelitis simulating bone tumours and other diseases (*discussion*), 407.
Whiston, T. B., and Walmsley, R. Reaction of bone and tendon after tunnelling of bone and insertion of tendon, *377.
White, A. Chronic bone abscess (*discussion*), 404.
Whitehead, R. G. Oxidative phosphorylation and calcification, 155.
Widdowson, E. M. *See* Dickerson, J. W. T., and Widdowson, E. M.
Wiles, P. *Book review*, 174.
Sir Gordon Gordon-Taylor (*obituary*), 845.
Andrews, P. S., and Bremner, R. A. Chondromalacia of patella, *65.
Wilkinson, J. A., and Carter, C. Congenital dislocation of hip, 652, *669.
Williams, P. Congenital dislocation of hip, 410.
Discussions, 402, 404, 405.
Foot stabilisation, 402.
Resection of large osteoclastomata, 406.
Unusual uses of Küntscher nail, 406.
Wilson, J. N. Treatment of fractures of medial epicondyle of humerus, *778.
Wood, H. L.-C. *Book review*, 417.
Subacromial bursitis (*discussion*), 158.
Wouters, H. W. Treatment of sarcoma of femur by radiotherapy followed by excision of femur and turn-up-plasty of leg, 650.
Wynn Parry, C. B. Symposium on hand surgery: rehabilitation, 648.

*Indicates full article.

SUBJECT INDEX

A

Abdominal neuromyotomy for flexion and adduction spasticity of both hips, 398.
Abductor pollicis longus and extensor pollicis brevis tendons, anatomical variations affecting surgery of de Quervain's disease, *352.
Accidents. *See* Injuries.
Achilles tendon. *See* Tendo calcaneus.
Albers-Schönberg's disease. *See* Osteopetrosis.
America. *See* United States.
Amputations
toes, all, for clawing, *90; (*correspondence*), 412.
See also Cineplasty.
Aneurysmal bone cyst of humerus, 410.
Angioma. *See* Haemangioma.
Ankle injuries, radiography in forced inversion, *793.
Ankylosing spondylitis, knee changes in, *142.
Antibiotics, atypical appearances in osteomyelitis due to, 407.
Arm. *See* Limbs, upper. *See also* Forearm.
Arteries
injuries accompanying brachial plexus injuries, 404.
See also Ischaemia; Vascular disorders.
Arthritis
deformans. *See* Osteoarthritis.
due to *Bacterium mycoides*, 858.
neuropathic, in diabetes mellitus, *769.
pyogenic
chronic, caused by foreign bodies, 405.
hip, in infancy, *11, 403.
in infancy, *706.
rheumatoid
knee in, flexion contracture, *730.
toes in, claw, amputation of all toes for, *90; (*correspondence*), 412.
tuberculous. *See* Tuberculosis, bone and joint.
Arthrochalasis multiplex congenita. *See* Joints, laxity, familial.
Arthrodesis. *See under names of joints.*
Arthroplasty, 857.
skin, 403.
See also under names of joints.
Australian Orthopaedic Association, annual meetings, 1958, 400; 1959, 407.
Auto-immunisation in etiology of disease, 649.
Automobile injuries, prevention, 862.
Autoradiography with tritiated thymidine in study of endochondral ossification, *824, 857.
Axis vertebra, odontoid process of, fracture, with anterior dislocation, 410.

B

Back
pain, low, 859.
stiff, in children, 651.
See also Spine; Intervertebral discs.
Bacterium mycoides arthritis of knee, 858.
Batchelor's pseudarthrosis of hip, results, *177.
Bed frame overhead, for crippled patient, 159.
Biceps brachii cineplasty, 407.
Biceps femoris, glomus tumour of, 858.
Birth palsy, rehabilitation in, 158.
Blood clotting inhibition in etiology of non-union of fractures of femoral neck, *226.
Blood vessels
tumours. *See* Glomus tumour; Haemangioma; Haemangiopericytoma.
See also Arteries; Ischaemia; Vascular disorders.
Bone and bones
blood supply
adjacent to epiphysial cartilage
effect of interruption of, *571.
electron microscope studies, *367.
injection studies, *97.
in occlusive vascular disease, changes in, *110.
calcification
in vitro, phosphatase activity in, *137.
mechanism of, 154.
role of citrate in, 855.
chemical composition, correlation with structure, 856.
collagen fibres, arrangement in human secondary osteones, *588.
congenital disorders
undiagnosed case, 652.
See also more specific terms.
cysts
aneurysmal, of humerus, 410.
multilocular, of femoral neck, 400.
simple, of humerus, 658.
disappearing. *See* Osteolysis, massive.
diseases. *See more specific terms.*
eosinophilic granuloma, 399.
fibroma, chondromyxoid, 405, *542.
fibrosarcoma simulating Paget's disease, 641.
form, mutability under mechanical stress, 153.
grafts and grafting, 653.
experimental, at Otago University, 659.
foetal bone, experimental study, 653.
for non-union of fractures, 653, 662.
preservation in ether, clinical results, 862.
growth
and development in rats, effect of size of litter, 156.
and remodelling, autoradiographic study, 396.
longitudinal
cell division in, autoradiographic study using tritiated thymidine, *824, 857.
stimulation
by insertion of bone or ivory pegs, *489.
for leg length discrepancy, 398.
tetracyclines in study of, 856.
See also Epiphysial cartilage; Osteogenesis.
haemangioma, 857.
marble. *See* Osteopetrosis.
mast cell infiltration of, 641.
matrix (*symposium*), 853.
metabolism, radioactive isotopes as indicators of, 397.
microradiography in diagnosis, 663.
necrosis, avascular
changes in radiographic density associated with, *626.
diagnosis, radioactive isotopes in, 650.
Paget's disease
chondrosarcoma in, 400.
diagnosis, differential, *641.
fracture of femoral neck in, 406.
histological abnormalities of bone matrix in, 854.
osteogenic sarcoma of calcaneum in, 400.
radiation injury, 410.
radioactive materials and (*symposium*), 394.
sarcoma
reticulum-cell, of femur, 650.
See also more specific terms.
structure
correlation with chemical composition, 856.
variations with age, 856.

* Indicates full article or appreciable part.

Bone and bones
synovioma, malignant, congenital, *742.
tuberculosis in the Bantu, problems of, 861.
tumours
experimental production using radioactive isotopes, dose response relationships, 394, 395.
giant-cell. *See* Osteoclastoma.
resection of, 406.
See also more specific terms.
yaws involving, tumoural gummatous, *785.
See also Epiphyses; Epiphysial cartilage; *and names of bones.*
Bone and Tooth Society, meetings, 1959, 154, 156; 1960, 394, 853, 855.
Book reviews:
Adams, J. C. Outline of orthopaedics, 3rd edition, 1960, 875.
Albert, F., and Medawar, P. B., *eds.* Biological problems of grafting, 1959, 872.
American Academy of Orthopaedic Surgeons Instructional Course Lectures, Vol. 16, 1959, 872.
Anderson, M. H., Bechtol, C. O., and Sollars, R. E. Clinical prosthetics for physicians and therapists, 1959, 414.
Baruffaldi, O., and Divano, N. La spina bifida, 1959, 172.
Bechtol, C. O., Ferguson, A. B., Jun., and Laing, P. G. Metals and engineering in bone and joint surgery, 1959, 865.
Bloch, B. Bonding of fractures by plastic adhesives, 875.
Borgström, S., Gelin, L.-E., and Zederfeldt, B. Formation of vein thrombi following tissue injury, 1959, 874.
Bowesman, C. Surgery and clinical pathology in the tropics, 1960, 873.
British Medical Bulletin, Vol. 16, No. 1, Antibiotics in medicine, 1960, 420.
Clarke, R., Badger, F. G., and Sevitt, S., *eds.* Modern trends in accident surgery and medicine, 1959, 867.
Cosentino, R. Atlas of anatomy and surgical approaches in orthopaedic surgery—Upper extremity, 1960, 871.
Duchenne, G. B. Physiology of motion, 1959, 664.
DuVries, H. L. Surgery of the foot, 1959, 417.
Flatt, A. E. Care of minor hand injuries, 1959, 865.
Goldstein, L. A. Surgical treatment of scoliosis, 1959, 869.
Greenfield, J. G., *et al.* Neuropathology, 1958, 415.
Greulich, W. W., and Pyle, S. I. Radiographic atlas of skeletal development of the hand and wrist, 2nd edition, 1959, 171.
Gruca, A. Chirurgia ortopedyczna, Vol. 1, 1959, 666.
Guillain, G. J.-M. Charcot 1825-1893: his life—his work, 1959, 420.
Gurdjian, E. S., and Webster, J. E. Head injuries, 1958, 167.
Guthrie, D., *et al.* The Royal Edinburgh Hospital for Sick Children, 1860-1960, 1960, 875.
Hampton, O. P., Jun., and Fitts, W. T., Jun. Open reduction of common fractures, 1959, 168.
Heikel, H. V. A. Aplasia and hypoplasia of the radius, 1959, 419.
Hohmann, G., Hackenbroch, M., and Lindemann, K., *eds.* Handbuch der Orthopädie, Bd. 3, 1959, 413.
Iselin, M. Chirurgie der Hand: Atlas der Operationstechnik, 1959, 174.
Jacobs, A. L. Arterial embolism in the limbs, 1959, 163.
Jamieson, R. A., and Kay, A. W. Textbook of surgical physiology, 1959, 415.
Book Reviews (*continued*):
Jokl, E. Clinical physiology of physical fitness and rehabilitation, 1958, 665.
Kivilaakso, R. The Schanz osteotomy for irreducible dislocation of the hip, 1959, 173.
Lange, F. Ein Leben für die Orthopädie, 1959, 421.
Langeron, P., and Croccel, L. Le phénomène de Raynaud, 1960, 874.
LeFanu, W. Catalogue of the portraits . . . in the Royal College of Surgeons of England, 1960, 667.
Leikkonen, O. Low back pain and sciatica, 1959, 173.
Lidström, A. Fractures of the distal end of the radius, 1959, 666.
Lindström, B. L. Function test for peripheral arterial circulatory insufficiency in the lower extremities in obstructive arterial disease, 1959, 874.
Lusted, L. B., and Keats, T. E. Atlas of roentgenographic measurement, 1959, 171.
McGregor, I. A. Fundamental techniques of plastic surgery, 1960, 665.
Merle d'Aubigné, R., and Ramadier, J.-O. Traumatismes anciens: rachis—membre inférieur, 1959, 170.
Merle d'Aubigné, R., *et al.* Actualités de chirurgie orthopédique et réparatrice . . . 1958-1959, 1959, 169.
Middlemiss, H. Radiology as a diagnostic aid in clinical surgery, 1960, 871.
Morrison, L. R. The effect of advancing age upon the human spinal cord, 1959, 873.
Niederecker, K. Der Plattfuss, 1959, 417.
Ospedali d'Italia Chirurgia, Vol. 1, No. 1, Nov. 1959, 667.
Reconstruction surgery and traumatology, Vol. 5, 1960, 168.
Roaf, R., Kirkaldy-Willis, W. H., and Cathro, A. J. M. Surgical treatment of bone and joint tuberculosis, 1959, 163.
Rose and Carless manual of surgery, 19th edition, 1960, 876.
Rusk, H. A., *et al.* Rehabilitation medicine, 1958, 871.
Schmorl, G., and Junghanns, H. The human spine in health and disease, 1st American edition, 1959, 420.
Sèze, S. de, Ryckewaert, A., and Maitre, M. L'épaule en pratique rhumatologique, 1959, 418.
Simon, G. Principles of bone X-ray diagnosis, 1960, 870.
Solonen, K. A. The sacroiliac joint, 1957, 419.
Souttar, Sir H., and Goligher, J. C., *eds.* Textbook of British surgery, Vol. 4, 1959, 165.
Steindler, A. Lectures on the interpretation of pain in orthopaedic practice, 1959, 413.
Stoddard, A. Manual of osteopathic technique, 1959, 174.
Surgery in World War II:
Hand surgery, 1955, 664.
Neurosurgery, Vol. 1, 1958, 166.
Physiologic effects of wounds, 1952, 165.
Terrier, J. C., *ed.* Beitrage zur manuellen Therapie, 1959, 174.
Torppi, P. Über Frakturen am oberen Humerusende 1959, 168.
Trifaud, A., and Bureau, H. Tumeurs bénignes des os, 1959, 170.
Turek, S. L. Orthopaedics, 1959, 864.
Vieten, H., Oberdalhoff, H., and Karcher, H. Klinische Röntgendiagnostik chirurgischer Erkrankungen, 1959, 416.
Virtama, P. Determination of the mineral content of human finger bones by silver analysis of roentgenograms, 1957, 171.

* Indicates full article or appreciable part.

Book reviews (*continued*):
Wiles, P. Essentials of orthopaedics, 3rd edition, 1959, 665.
Wiles, P. Fractures, dislocations and sprains, 1960, 866.
Wilkinson, A. W. Body fluids in surgery, 1960, 876.
Brachial plexus injuries, 658.
birth, rehabilitation in, 158.
traction, partial, arthrodesis of shoulder for, 409.
with vascular damage, 404.
Brain. *See* Cerebral aneurysm; Cerebral palsy; Encephalitis; Epilepsy.
Brazilian Society for Surgery of the Hand, foundation of, 411.
British Council for Rehabilitation, Dame Georgiana Buller Memorial Lecture, 1960, 852.
British Orthopaedic Association
amendment of closing date for receipt of papers for scientific meetings, 411.
election of fellows, etc., 654.
meetings, 1960, spring, 646; future, 863.
Robert Jones Golf Cup competition, 1960, 654.
Travelling Scholarships, report of scholar for 1959, 655.
Browne, Denis, treatment of congenital dislocation of hip, results, 401.
Buller, Dame Georgiana, Memorial Lecture, 1960, 852.
Bursitis, subacromial, 158.

C

Calcaneal tendon. *See* Tendo calcaneus.
Calcaneum
fractures, arthrodesis for, talo-calcanean, primary, *336.
osteotomy, for recurrent or resistant club foot, 858.
Paget's disease, osteogenic sarcoma in, 400.
Calcification
in vitro, phosphatase activity in, *137.
mechanism of, 154.
of articular cartilage, associated with hyperparathyroidism, *530.
role of citrate in, 855.
Calf, ischaemia, localised, 659.
Calvé-Legg-Perthes' disease. *See* Hip, osteochondritis.
Canada, orthopaedics in, report of Travelling Fellows, 1960, 847.
Canute, his laws of compensation for loss of fingers, *390.
Carpo-metacarpal joints
dislocation, *772.
of thumb, osteoarthritis, excision of trapezium for, *502.
Cartilage
articular
calcification of, associated with hyperparathyroidism, *530.
degeneration of, 854.
epiphysial
blood supply adjacent to
effect of interruption of, *571.
influence on growth
electron microscope studies, *367.
injection studies, *97.
calcification
in vitro, phosphatase activity in, *137.
mechanism of, 154.
See also Bone and bones, growth, longitudinal; Osteogenesis; Epiphysiodesis.
semilunar. *See* Menisci.
tumours. *See* Chondroblastoma; Chondromyxoid fibroma; Chondrosarcoma; Dyschondroplasia.
Cerebral aneurysm, anterior, occlusion of, rehabilitation after, 158.
Cerebral palsy, recent advances in, 861.
Cervical spondylosis, 860.
hyperextension injury following, with rupture of oesophagus, *356.
Charcot's arthropathy in diabetes mellitus, *769.
Charnley, John, arthrodesis of hip by central dislocation, *466, *476, 659.
Chemotherapy. *See* Antibiotics.
Chest injuries, crush, management of flail anterior segment, 652.
Chondroblastoma, 409.
Chondromalacia patellae, excision of articular cartilage for, follow-up, *65.
Chondromyxoid fibroma of bone, *542.
humerus, head of, 405.
Chondrosarcoma, 409.
in Paget's disease, 400.
Chordoma
of first lumbar vertebra, *565.
sacral, metastasising, *556.
thoracic, *560.
Cineplasty, biceps brachii, 407.
Citrate, influence on composition and form of calcium phosphate precipitates, 855.
Clavicle, fractures
compound, non-union of, 863.
non-union of, 406.
Claw foot, treatment, excision of head and neck of metatarsals, 409.
Claw toes
amputation of all toes for, *90; (*correspondence*), 412.
due to sciatic nerve injury, 224.
reconstruction of forefoot for, 399; (*correspondence*), 412.
Club foot
recurrent or resistant, treatment
medial and posterior release operation, 402.
osteotomy of calcaneum, 858.
treatment
arthrodesis, Lambrinudi's, modified, *333.
soft-tissue operation, 407.
Collagen of bone
arrangement of fibres in human secondary osteones, *588.
changes in osteoporosis, 855.
Colles's fracture
fixation, posterior plaster slab, 159.
injury of articular disc associated with, 522.
Colonna, Paul C.
capsular arthroplasty of hip, for congenital dislocation, 701.
trochanteric reconstruction operation, *5.
Compound palmar ganglion. *See* Tuberculous tenosynovitis.
Congenital abnormalities
multiple, 405, 410.
undiagnosed case, 652.
See also under parts involved.
Connective tissue matrix, electron microscope study, 853.
Convulsions, injuries due to,
dislocation, posterior, of both shoulders, *728.
fracture of femoral neck, bilateral, 236.
Coxa plana. *See* Hip, osteochondritis.
Coxa vara
adolescent. *See* Slipped upper femoral epiphysis.
congenital, 404, 857.
etiology, interruption of blood supply to femoral head, *265.
Cysts
bone
aneurysmal, of humerus, 410.
multilocular, of femoral neck, 400.
simple, of humerus, 658.

* Indicates full article or appreciable part.

Cysts
dermoid, inclusion, following lumbar puncture, 651.
synovial
of fingers, 399.
See also Ganglia.
Cytoma, plasma cell. *See* Myeloma.

D

Danish Orthopaedic Association, meetings, 1959, 660.
De Quervain's disease, anatomical variations affecting surgery of, *352.
Deformities
congenital, multiple, 405, 410.
undiagnosed case, 652.
See also under parts involved.
Deltoid muscle, fibrosarcoma, 409.
Dermoids, inclusion, following lumbar puncture, 651.
Diabetes mellitus, Charcot's arthropathy in, *769.
Discography, 403.
Discs, intervertebral
cervical. *See* Cervical spondylosis.
infection in children, 410, 651.
lesions
chemistry of, 855.
in adolescents, 651.
treatment, operative, results, 860.
See also Back pain, low.
Dislocations. *See under names of joints.*
Drop foot
arthrodesis for, Lambrinudi's, modified, *333.
due to sciatic nerve injury, reconstruction operations for, 221.
transplantation of tibialis posterior for, 410.
Dutch Orthopaedic Society. *See* Nederlandse Orthopaedische Vereniging.
Dyschondroplasia, Ollier's, case followed for 54 years, *344.
Dysplasia epiphysialis
capitis femoris, 663.
multiplex, 862.
confined to lower limbs, *313.

E

East Anglian Orthopaedic Club meetings, 1959, 159, 399; 1960, 658, 857.
Edinburgh
orthopaedic pioneers of, 393.
Royal College of Surgeons, meeting of British Editorial Board of *Journal* at, presentation of gavel by Sir Walter Mercer, 154.
Elbow
arthritis, nonspecific, 410.
dislocation
lateral
of head of radius, with greenstick fracture of upper end of ulna, *782.
with fracture of medial epicondyle of humerus, 778.
recurrent, 399, 406.
fracture-dislocation, 858.
Monteggia, with unreduced radial head, 406.
injuries in javelin throwers, *788
tennis, resection of synovial membrane for, 400.
Electroconvulsive therapy, hip injuries during, bilateral, *236.
Enamel, dental, organic matrix of
in some diseases, 854.
quantitative changes during growth, 853.
Encephalitis, recurrent posterior dislocation of shoulder after, 863.
Enchondroma, multiple. *See* Ollier's dyschondroplasia.
Eosinophilic granuloma of bone, 399.
Epilepsy, injuries due to convulsions
dislocation, posterior of both shoulders, *728.
fracture of femoral neck, bilateral, 236.
Epiphyses
displacement in spina bifida, *387.
See also under names of bones.
See also Osteochondritis.
Epiphysial cartilage
blood supply adjacent to
effect of interruption of, *571.
influence on growth
electron microscope studies, *367.
injection studies, *97.
calcification
in vitro, phosphatase activity in, *137.
mechanism of, 154.
See also Bone and bones, growth, longitudinal; Osteogenesis.
Epiphysial dysplasia, multiple, 862.
confined to lower limbs, *313.
Epiphysiodesis for scoliosis, *51.
Epiphysitis. *See* Osteochondritis.
Europe, orthopaedics in, report of British Orthopaedic Association travelling scholar for 1959, 655.
Extensor pollicis brevis and abductor pollicis longus tendons, anatomical variations affecting surgery of de Quervain's disease, *352.
Extremities. *See* Limbs.

F

Facial nerve paralysis in osteopetrosis, 303.
Femur
epiphysis, upper
osteochondritis. *See* Hip, osteochondritis.
slipping of
bilateral, 863.
pathogenesis, new concept, *273
treatment, *289.
Moore's pins, extractor for, *570.
surgical, *280.
excision
and turn-up plasty of leg, for reticulum-cell sarcoma, 650.
of head and neck, results, *177.
fractures
neck
analysis of forces producing, *633.
avascular necrosis after, 650.
anatomical study, *802.
diagnosis
radioactive isotopes in, 650.
radioactive phosphorus uptake, *21.
bilateral, *236.
in Paget's disease, 406.
non-union of
Colonna's trochanteric reconstruction operation for, *5.
etiology, *226.
treatment
arthroplasty, Thompson's prosthesis, 857.
nailing, effect on blood supply of head and neck, *794.
shaft
due to myeloma, 409.
nailing of, intramedullary, Küntscher, 658.
stiff knee complicating, quadricepsplasty for, 159.
with head injury, treatment, *425.
stress, longitudinal, *508.
supracondylar, non-union of, bone grafting for, 409.

* Indicates full article or appreciable part.

Femur
growth at upper end, normal and abnormal, *264.
head, necrosis
after fractures of femoral neck, 650.
anatomical study, *802.
etiology, nailing, *794.
changes in radiographic density associated with, *626.
diagnosis
radioactive isotopes in, 650.
radioactive phosphorus uptake, *11.
in congenital dislocation of hip, 701.
See also Hip, osteochondritis.
neck, anteversion, 659.
in congenital dislocation of hip, 687, 693, 699.
osteomyelitis of upper end in infancy, *11.
osteotomy
displacement, results, *177.
for deformities of neck in congenital dislocation of hip, 699.
intertrochanteric
fixation for, nail-plate, McKee's, 858.
for osteoarthritis of hip, arrest of disease process due to (*editorial*), 423.
McMurray's, *480.
fixation for, internal, 398.
supracondylar, in children, nature of remodelling after, 653.
sarcoma
osteogenic, 658.
reticulum-cell, 650.
short, congenital, 404.
torsion. *See* Femur, neck, anteversion.
Fibrogenesis imperfecta, histological abnormalities of bone matrix in, 854.
Fibroma, chondromyxoid, of bone, 405, *542.
Fibrosarcoma
of bone simulating Paget's disease, 641.
of deltoid muscle, 409.
Fingers
compensation, and King Canute, *390.
cysts, synovial, 399.
locking of middle, *75.
See also Thumb.
See also Polydactyly.
Flat foot, painful, movement of big toe in, 159.
Foot
arthrodesis. *See* Tarsus, arthrodesis.
calcaneus, in poliomyelitis, treatment, 861.
Charcot's arthropathy, in diabetes mellitus, *769.
claw, treatment, excision of head and neck of metatarsals, 409.
club
recurrent or resistant, treatment
medial and posterior release operation, 402.
osteotomy of calcaneum, 858.
treatment
arthrodesis, Lambrinudi's, modified, *333.
soft-tissue operation, 407.
drop
arthrodesis for, Lambrinudi's, modified, *333.
due to sciatic nerve injury, reconstruction operations for, 221.
transplantation of tibialis posterior for, 410.
osteolysis, massive, follow-up for thirty years (*correspondence*), 162.
See also Ankle; Tarsus; Toes.
See also Metatarsalgia.
Forearm
osteoclastoma, excision and bone grafting for, 400.
rotation, anatomy of inferior radio-ulnar joint in relation to, 518.
ulnar dimelia, *549.
Foreign bodies, chronic septic arthritis caused by, 405.
Fractures
bonding with plastic adhesives, 407.
intra-articular, non-union of, etiology, *226.
nailing of, intramedullary, 398.
Küntscher, 406.
femur, 658.
non-union of, treatment
bone grafting, 653, 662.
" petal " technique, experimental study, *126.
surgical (*editorial*), 3.
stress, 405.
longitudinal, *508.
patella, *71.
sesamoid bones of big toe, *840.
treatment in Vienna at Workmen's Accident Hospital, 656.
See also under names of bones.
Fragilitas ossium. *See* Osteogenesis imperfecta.
French Orthopaedic Society. *See* Société Française d'Orthopédie et de Traumatologie.

G

Gallie's arthrodesis of talo-calcanean joint for fractures of calcaneum, *336.
Ganglia
compound palmar. *See* Tuberculous tenosynovitis.
intraneural, of lateral popliteal nerve, 652.
See also Cysts, synovial.
Giant-cell tumour of bone. *See* Osteoclastoma.
Gillies, Sir H., method of thumb lengthening, 445.
Glomus tumour of biceps femoris, 858.
Gordon-Taylor, Sir Gordon (*obituary*), 845.
Gorham's disease. *See* Osteolysis, massive.
Granuloma, eosinophilic, of bone, 399.
Growth and skeletal development in rats, effect of size of litter, 156.

H

Haemangioma
knee, 857.
synovial cavernous, 159.
spine, 857.
Haemangiopericytoma, orthopaedic manifestations, *80.
Haemarthrosis, blood clotting inhibition in, in etiology of non-union of fractures of femoral neck, *226.
Hallux. *See* Toes, big.
Hallux rigidus, degeneration of sesamoid bones in, 158.
Hallux valgus
degeneration of sesamoid bones in, 158.
in adolescence
and early adult life, natural history of, *749.
treatment, operative, *761.
treatment, metatarsal osteotomy with excision of base of phalanx, 158.
Hand
fractures, treatment, operative, 647.
injuries
major, prognosis, functional and economic, 648.
mutilating, treatment, 648.
rehabilitation after, 648.
treatment, organisation of, 648.
nerves, injuries, repair, primary, 408.
sensibility, restoration by neurovascular island flap, 647.
skin grafting in, 646
surgery, history of, 646.
tendons
flexor, injuries, repair, 647.
injuries, 158.
repair, primary, 408, 647.
See also names of tendons.

* Indicates full article or appreciable part.

Hand
See also Wrist; Fingers; Thumb.
See also Polydactyly.
Hand Surgeons' Club of Great Britain, joint meeting with Second Hand Club and British Orthopaedic Association, 1960, April, 646.
Hashimoto's disease, 649.
Head injuries with fracture of femoral shaft, treatment, *425.
Hemiplegia, adult, 158.
Hip
approach to, low posterior, 408.
arthritis, pyogenic, in infancy, *11, 403, *706.
arthrodesis, central dislocation operation of Charnley, *466, *476, 659.
arthroplasty
acrylic, after excision of femur and turn-up plasty of leg, 650.
Colonna's capsular, for congenital dislocation, 701.
cup, for osteoarthritis, results, 403.
Judet, results, 660.
replacement
anchorage of prosthesis with acrylic cement, *28.
for fracture of femoral neck in Paget's disease, 406.
Thompson's prosthesis, for fractures of femoral neck, 857.
conditions, unusual, 399.
dislocation
congenital
bilateral, 862.
prognosis, measurement of acetabular angle of opposite hip, 652, *669.
spontaneous re-position of, 858.
treatment, 410, *689.
at Istituto Rizzoli, 656.
conservative, 401.
results, 652, *669.
results, comparison of frame and manipulative reduction, *253.
surgical, 400.
traumatic
accompanying multiple injuries, late discovery of, 857.
irreducible by closed manipulation, 858.
flexion deformity, treatment, 863.
injuries, bilateral, *236.
osteoarthritis, treatment
arthrodesis, central dislocation operation of Charnley, *466, *476, 659.
arthroplasty, cup, results, 403.
operative, 659.
osteotomy of femur
arrest of disease process due to (*editorial*), 423.
McMurray's, *480.
reconstruction operations, results, *177.
shelf operation, 653.
spinothalamic tractotomy, 409.
osteochondritis
diagnosis, differential, dysplasia epiphysialis capitis femoris, 663.
etiology, changes in ligamentum teres, 651.
following congenital dislocation, incidence, effect of mode of reduction on, *253.
treatment, ambulant, follow-up, 661.
paralysis, spastic, flexion and adduction, bilateral, abdominal neuromyotomy for, 398.
shelf operation for osteoarthritis, 653.
surgery, reconstruction operations
Colonna's trochanteric, *5.
results, further review, *177.
See also Coxa vara.
Histiocytosis. *See* Eosinophilic granuloma.
Hodgkin's disease, bone changes in, 641.
Hôpital Cochin, opening of new block for orthopaedics and injuries, 159.
Hospitals. *See names of hospitals.*
Humerus
cyst, simple, 658.
fractures, epicondyle
lateral, non-union of, etiology, 233.
medial, 858.
treatment, *778.
synovioma, malignant, congenital, *742.
tumours of head of, 405.
Hyperparathyroidism
calcification of articular cartilage associated with, *530.
secondary, *See* Renal osteodystrophy.
Hypervitaminosis D rickets, 606.
Hypothyroidism. *See* Hashimoto's disease.

I

Ilio-psoas myotomy with obturator neurectomy for flexion and adduction spasticity of both hips, 398.
Implantation dermoids following lumbar puncture, 651.
Infantile paralysis. *See* Poliomyelitis.
Injuries
multiple, fracture of femoral shaft with head injury, treatment, *425.
road, prevention of, 862.
soft-tissue, assessment of tissue viability in, intravenous dye technique, 646.
See also Fractures *etc., and under parts involved.*
Interspinous ligament, rupture of, 403.
Intervertebral discs
cervical. *See* Cervical spondylosis.
infection in children, 410, 651.
lesions
chemistry of, 855.
in adolescents, 651.
treatment, operative, results, 860.
See also Back pain, low.
See also Discography.
Intramedullary nailing, 398.
hypertrophic bone reaction after, extreme, 409.
Küntscher
femur, 658.
unusual applications, 406.
Irish Orthopaedic Club, meeting, 1960, May, 858.
Irwin, Charles Gordon (*obituary*), 148.
Ischaemia, localised, of calf, 659.
Isotopes, radioactive
and the skeleton (*symposium*), 394.
in diagnosis of avascular necrosis of bone, 650.
See also Autoradiography.
See also Phosphorus; Strontium.
Italian Orthopaedic Society. *See* Società Italiana di Ortopedia e Traumatologia.

J

Javelin thrower's elbow, *788.
Joints
cartilage, articular
calcification of, associated with hyperparathyroidism, *530.
degeneration of, 854.
Charcot, in diabetes mellitus, *769.
cysts, synovial
of fingers, 399.
See also Ganglia.
fractures, non-union of, etiology, *226.
haemangioma of knee, 857.
synovial cavernous, 159.

* Indicates full article or appreciable part.

Joints
laxity, familial, association with recurrent dislocation of patella and of shoulder, *721.
synovioma, malignant, 650.
tuberculosis. *See* Bone and bones, tuberculosis.
See also Arthritis; Arthroplasty; *and names of joints.*
Jones, Lady, Lecture, Liverpool University, 1959, 153.
Jones, Sir Robert
Golf Cup competition, 1960, 654.
Lecture, Royal College of Surgeons of England, 1959, 393.
Journal of Bone and Joint Surgery, British Editorial Board meeting at Royal College of Surgeons of Edinburgh, presentation of gavel by Sir Walter Mercer, 154.
Judet arthroplasty of hip, results, 177, 660.
Juxta-cortical osteogenic sarcoma of head of humerus, 405.

K

Kidneys. *See* Renal osteodystrophy.
Knee
approach to lateral compartment, lateral, direct, *348.
arthritis, pyogenic, in infancy, *706.
arthroplasty with hinged prosthesis, *31.
deformities in poliomyelitis, supracondylar osteotomy for, nature of remodelling after, 653.
dislocation
congenital, anterior, bilateral, with associated deformities, 410.
postero-lateral, irreducible, 409.
flexion contracture, *730.
haemangioma, 857.
synovial cavernous, 159.
in ankylosing spondylitis and Reiter's disease, *142.
meniscectomy, osteoarthritis after, 408.
menisci, cysts of lateral
excision of, approach for, lateral, direct, *348.
radiological diagnosis, *144.
osteochondritis dissecans, 858.
bilateral, 405, 410.
stiff, post-traumatic, quadricepsplasty for, 159.
tuberculosis, 409.
See also Patella.
Küntscher's intramedullary nailing
femur, 658.
unusual applications, 406.
Kyphosis, growth of spine in, and its mechanical control, *44.

L

Lahz, John Rudolph Sergius (*obituary*), 644.
Lambrinudi's arthrodesis for drop foot
due to sciatic nerve injury, 221.
modified, *333.
Lateral popliteal nerve, ganglia of, intraneural, 652.
Leg
ischaemia, localised, 659.
See also Limbs, lower.
Legg-Calvé-Perthes' disease. *See* Hip, osteochondritis.
Léri type of osteopetrosis. *See* Melorheostosis.
Ligaments. *See names of ligaments.*
Ligamentum teres, changes in, in etiology of osteochondritis of hip, 651.
Limbs
lower
dysplasia, epiphysial, familial, *313.
ischaemia, localised, of calf, 659.
length equalisation, growth stimulation, 398.
by insertion of bone or ivory pegs, *489.
Limbs
upper
congenital deformities, ulnar dimelia, *549.
paralysis, complete, mechanical aid for, *358.
prostheses, pneumatic, 657.
See also Forearm.
Liverpool University
degree of M.Ch.Orth., examination results, 1959, 153.
Lady Jones lecture, 1959, 153.
Los Angeles, Rancho los Amigos, 850.
Lovelace Clinic, expansion of staff at, 411, 863.
Lumbar puncture, complications, implantation dermoids, 651.
Lymphadenoma. *See* Hodgkin's disease.

M

McIndoe, Sir Archibald Hector (*obituary*), 844.
McKee's nail-plate for fixation after intertrochanteric osteotomy, 858.
McMurray's osteotomy, *480.
fixation for, internal, 398.
for osteoarthritis of hip, arrest of disease process due to (*editorial*), 423.
results, *177.
Marble bones. *See* Osteopetrosis.
Mast cells, infiltration of bones by, 641.
Median nerve and ulnar nerve injuries at wrist, repair of, 647.
Medicine
diagnosis and treatment in, new approach to, 649.
specialism in, 153.
Medullary nailing. *See* Intramedullary nailing.
Melorheostosis, 857.
Meniscectomy, osteoarthritis after, 408.
Menisci, cysts of lateral
excision of, approach for, lateral, direct, *348.
radiological diagnosis, *144.
Mercer, Sir Walter, presentation of gavel to British Editorial Board of *Journal* by, 154.
Merle d'Aubigné, R., decoration with Order of Officer of Legion of Honour, 159.
Mesenchymoma of popliteal fossa, 159.
Metacarpo-phalangeal joint of middle finger, locking of, *75.
Metals, implants of. *See* Steel, stainless.
Metatarsalgia, Morton's, 403.
Microradiography in diagnosis of bone disease, 663.
Miller, Alexander (*obituary*), 150.
Monteggia fracture-dislocation with unreduced radial head, 406.
Moore's pins, extractor for, *570.
Morton's metatarsalgia, 403.
Mucopolysaccharides of bone matrix, 853, 854.
Multiple enchondroma. *See* Ollier's dyschondroplasia.
Multiple epiphysial dysplasia 862.
confined to lower limbs, *313.
Muscles
ischaemia, localised, of calf, 659.
See also names of muscles.
"Museum pages," *142, *387, *641, *840.
Myeloma
fracture of femur due to, 409.
multiple, 407.
Myxoedema developing from Hashimoto's disease, 649.

N

Nailing, intramedullary, 398.
hypertrophic bone reaction after, extreme, 409.
Küntscher
of femur, 658.
unusual applications, 406.

* Indicates full article or appreciable part.

Nail-plates
for fixation after McMurray's osteotomy, 398.
McKee's, for fixation after intertrochanteric osteotomy, 858.
Navicular bone (carpal). *See* Scaphoid bone.
Nederlandse Orthopaedische Vereniging, joint meeting with British Orthopaedic Association, 1960, April, 646.
Neoplasms. *See* Tumours.
Nerves
facial, paralysis in osteopetrosis, 303.
injuries in hand, repair, primary, 408.
lateral popliteal, ganglia of, intraneural, 652.
median and ulnar, injuries at wrist, repair of, 647.
obturator, section of, with iliopsoas myotomy, for flexion and adduction spasticity of both hips, 398.
plantar digital, neuroma of, 403.
radial, lesion in lateral dislocation of radio-humeral joint with greenstick fracture of upper end of ulna, 782.
sciatic
injuries (*editorial*), 175.
late consequences, *213.
results of repair, *205.
paralysis, neonatal, due to injection into umbilical cord, *736.
sympathetic. *See* Sympathectomy.
ulnar
and median, injuries at wrist, repair of, 647.
lesion in javelin thrower, 788.
lesions in fractures of medial epicondyle of humerus, 780.
Netherlands Orthopaedic Society. *See* Nederlandse Orthopaedische Vereniging.
Neuroma, plantar digital, 403.
Neuromyotomy, abdominal, for flexion and adduction spasticity of both hips, 398.
Neuropathic arthritis in diabetes mellitus, *769.
New Zealand Orthopaedic Association, annual meeting, 1959, 659.
Nursing hoists, over-bed frame for use by patient, 159.

O

Obituaries:
Gordon-Taylor, Sir Gordon, 845.
Irwin, Charles Gordon, 148.
Lahz, John Rudolph Sergius, 644.
McIndoe, Sir Archibald Hector, 844.
Miller, Alexander, 150.
Obstetrical paralysis, rehabilitation in, 158.
Obturator neurectomy with iliopsoas myotomy for flexion and adduction spasticity of both hips, 398.
Odontoid process of axis vertebra, fracture, with anterior dislocation, 410.
Oesophagus, rupture accompanying hyperextension injury of cervical spine, *356.
Ollier's dyschondroplasia, case followed for 54 years, *344.
Orthopaedic surgeons, Edinburgh pioneers, 393.
Orthopaedics
in Europe, report of British Orthopaedic Association travelling scholar for 1959, 655.
in rural areas, problems of, 858.
in United States and Canada, report of Travelling Fellows, 1960, 847.
Os calcis. *See* Calcaneum.
Ossification. *See* Osteogenesis.
Osteitis deformans. *See* Paget's disease of bone.
Osteoarthritis
carpo-metacarpal joint of thumb, excision of trapezium for, *502.
etiology and pathogenesis, 854.
Osteoarthritis
hip, treatment
arthrodesis, central dislocation operation of Charnley, *466, *476, 659.
arthroplasty, cup, results, 403.
operative, 659.
osteotomy of femur
arrest of disease process due to (*editorial*), 423.
McMurray's, *480.
reconstruction operations, results, *177.
shelf operation, 653.
spinothalamic tractotomy, 409.
knee, after meniscectomy, 408.
metacarpo-phalangeal joint, in etiology of locked middle finger, *75.
spine, cervical. *See* Cervical spondylosis.
Osteochondritis
dissecans, knee, 858.
bilateral, 405, 410.
hip
diagnosis, differential, dysplasia epiphysialis capitis femoris, 663.
etiology, changes in ligamentum teres, 651.
following congenital dislocation, incidence, effect of mode of reduction on, *253.
treatment, ambulant, follow-up, 661.
sesamoid bones of big toe, *842.
spine. *See* Scheuermann's disease.
Osteoclastoma, 658.
femoral neck, causing fracture, 400.
forearm, excision and bone grafting for, 400.
resection of, 406.
Osteodystrophy, renal. *See* Renal osteodystrophy.
Osteogenesis
activation by "petal" technique, *126.
See also Bone and bones, growth; Cartilage, epiphysial.
Osteogenesis imperfecta, histological abnormalities of bone matrix in, 854.
Osteogenic sarcoma
femur, 658.
in Paget's disease of calcaneum, 400.
parosteal, of head of humerus, 405.
seasonal rhythm in presentation of, 156.
Osteolysis, massive, of foot, follow-up for thirty years (*correspondence*), 162.
Osteoma, parosteal, of head of humerus, 405.
Osteomyelitis
atypical appearances due to antibiotics, 407.
chronic, 404.
after compound fractures of tibia, 407.
diagnosis and treatment, *535.
femur, upper end, in infancy, *11.
spine, 660.
in children, *86.
See also Intervertebral discs, infection.
Osteopetrosis
avian, 855.
changing concept, *303.
familial, 858.
histological abnormalities of bone and cartilage matrix in, 854.
Léri type. *See* Melorheostosis.
pathogenesis, experimental study, *606.
Osteoporosis, changes in collagen in, 855.
Osteosarcoma. *See* Osteogenic sarcoma.
Osteosclerosis due to intermittent administration of large doses of vitamin D in rat, *606.
Osteotomy. *See under names of bones.*

P

Paget's disease of bone
chondrosarcoma in, 400.

* Indicates full article or appreciable part.

Paget's disease of bone
diagnosis, differential, *641.
fracture of femoral neck in, 406.
histological abnormalities of bone matrix in, 854.
osteogenic sarcoma of calcaneum in, 400.
Paralysis. *See* Birth palsy; Cerebral palsy; Hemiplegia; Paraplegia; Poliomyelitis; *and under parts involved.*
Paraplegia due to fracture-dislocation of thoracolumbar spine, 661.
Parathormone, effect on oxidative phosphorylation, 155.
Parathyroid glands. *See* Hyperparathyroidism.
Parosteal osteogenic sarcoma of head of humerus, 405.
Patella
bipartite, causing symptoms, 73.
chondromalacia, excision of articular cartilage for, follow-up, *65.
dislocation, recurrent, association with familial joint laxity, *721.
fractures, stress, *71.
Pelvis, fractures, diametric, *432.
Periosteal osteogenic sarcoma of head of humerus, 405.
Perthes' disease. *See* Hip, osteochondritis.
Pes cavus. *See* Claw foot.
Pes planus. *See* Flat foot.
Phosphatase, alkaline
activity in calcification *in vitro*, *137.
serum, raised levels in osteopetrosis, 303.
Phosphorus, radioactive, in diagnosis of avascular necrosis of femoral head, *21.
Plantar digital neuroma, 403.
Plasmacytoma. *See* Myeloma.
Plastics
anchorage of femoral head prostheses with acrylic cement, *28.
bonding of fractures with, 407.
Poliomyelitis
diagnosis, differential, osteomyelitis, 535.
foot in
calcaneus, treatment, 861.
drop, transplantation of tibialis posterior for, 410.
knee deformities in, supracondylar osteotomy for, nature of remodelling after, 653.
lower limbs in, length equalisation, growth stimulation by insertion of bone or ivory pegs, *489.
treatment at Rancho los Amigos, Los Angeles, 850.
Polysaccharides of bone matrix, 853, 854.
Polydactyly with ulnar dimelia, *549.
Popliteal nerve, lateral, ganglia of, intraneural, 652.
Posterior tibial muscle and tendon. *See* Tibialis posterior.
Pott's disease. *See* Spine, tuberculosis.
Pseudarthrosis, congenital, of tibia, 404.

Q

Quadricepsplasty, Thompson's, 159.
de Quervain's disease, anatomical variations affecting surgery of, *352.

R

Radial nerve lesion in lateral dislocation of radio-humeral joint with greenstick fracture of upper end of ulna, 782.
Radiation injury to bone, 410.
Radioactive isotopes
and the skeleton (*symposium*), 394.
in diagnosis of avascular necrosis of bone, 650.
See also Autoradiography.
See also Phosphorus; Strontium.
Radio-humeral joint. *See* Elbow.
Radio-ulnar joint, inferior
anatomy, functional, 518.
Radio-ulnar joint, inferior
dislocation
anterior, *515.
bilateral, 658.
injuries of articular disc, *522.
Radius
absence of, congenital, 857.
with ulnar dimelia, *549.
fracture, Colles's
fixation, posterior plaster slab, 159.
injury of articular disc associated with, 522.
head of, dislocation
lateral, with greenstick fracture of upper end of ulna, *782.
See also Monteggia fracture-dislocation.
Rancho los Amigos (Los Angeles), 850.
Rehabilitation
at Texas Institute for Rehabilitation and Research, 851.
pioneers of, 852.
Reiter's disease, knee changes in, *142.
Renal osteodystrophy (*symposium*), 156.
case with instability of legs, 404.
See also Rickets, resistant.
Reticulum-cell sarcoma of femur, 650.
Rheumatoid arthritis
knee in, flexion contracture, *730.
toes in, claw, amputation of all toes for, *90; (*correspondence*), 412.
Rickets
due to vitamin D excess, 606.
resistant, orthopaedic aspects, 409.
See also Renal osteodystrophy.
Road injuries, prevention, 862.
Rotator cuff injuries, 409.
Royal College of Surgeons of Edinburgh, meeting of British Editorial Board of *Journal* at, presentation of gavel by Sir Walter Mercer, 154.
Royal College of Surgeons of England
Robert Jones lecture, 1959, 393.
Watson-Jones lecture, 1959, 153.

S

Sacrum, chordoma of, metastasising, *556.
Sarcoma
osteogenic
femur, 658.
in Paget's disease of calcaneum, 400.
parosteal, of head of humerus, 405.
seasonal rhythm in presentation of, 156.
reticulum-cell, of femur, 650.
synovial. *See* Synovioma, malignant.
See also more specific terms.
Scaphoid bone (carpal), fractures, 160, 860.
delayed union of, 850.
non-union of, etiology, 233.
Scheuermann's disease, growth of spine in, and its mechanical control, 49.
Sciatic nerve
injuries (*editorial*), 175.
late consequences, *213.
results of repair, *205.
paralysis, neonatal, due to injection into umbilical cord, *736.
See also Lateral popliteal nerve.
Scoliosis
growth of spine in, and its mechanical control, *51.
treatment, 160.
in France, 655.
Second Hand Club, joint meeting with Hand Club and British Orthopaedic Association, 1960, April, 646.
Seddon, Herbert John, admitted "Doctor Honoris Causa of University of Grenoble," 411.

* Indicates full article or appreciable part.

Semilunar cartilages. *See* Menisci.
Sesamoid bones of big toe
degeneration in hallux valgus or rigidus, 158.
disorders, *840.
Sheffield Regional Orthopaedic Club meeting, 1960, March, 858.
Shoulder
approach to, axillary, 851.
arthrodesis for brachial plexus lesion, 409.
dislocation
posterior
bilateral, *728.
recurrent, following encephalitis, 863.
recurrent, association with familial joint laxity, *721.
rotator cuff injuries, 409.
See also Subacromial bursitis.
Skeleton. *See* Bone and bones.
Skin arthroplasty, 403.
Slipped upper femoral epiphysis
bilateral, 863.
pathogenesis, new concept, *273.
treatment, *289.
Moore's pins, extractor for, *570.
surgical, *280.
Società Italiana di Ortopedia e Traumatologia, annual meeting, 1959, 161.
Société Française d'Orthopédie et de Traumatologie, annual meeting, 1959, 159.
South African Orthopaedic Association, annual congress, 1959, 859.
South-East Metropolitan Regional Orthopaedic Club meeting, 1959, Nov., 158.
South-West Orthopaedic Club meeting, 1959, Nov., 397.
Spina bifida, epiphysial displacement in, *387.
Spine
approach to, transthoracic, 399.
arthrodesis
lumbo-sacral, follow-up, 660.
transthoracic, 406.
cervical
dislocation, 858.
fracture of odontoid process with anterior dislocation, 410.
injuries
hyperextension, with rupture of oesophagus, *356.
rupture of posterior ligament, 405.
See also Spondylosis, cervical.
chordoma
of first lumbar vertebra, *565.
sacral, metastasising, *556.
thoracic, *560.
curvature. *See* Kyphosis; Scoliosis.
fracture-dislocation, thoraco-lumbar, paraplegia due to, 661.
fractures, thoraco-lumbar, 662.
growth of, and its mechanical control, *40.
haemangioma, 857.
injuries, mechanics of, *810.
ligament, interspinous, rupture of, 403.
osteochondritis. *See* Scheuermann's disease.
osteomyelitis, 660.
in children, *86.
See also Intervertebral discs, infection.
surgery, 659.
tuberculosis, growth of spine in, and its mechanical control, 47.
See also Back; Intervertebral discs.
See also Spondylitis, ankylosing.
Spinothalamic tractotomy for osteoarthritis of hip, 409.
Splint, Thomas's, in treatment of fractures of femoral shaft associated with head injuries, 425.
Spondylarthritis. *See* Intervertebral discs, infection.
Spondylitis, ankylosing, knee changes in, *142.
Spondylosis, cervical, 860.
hyperextension injury following, with rupture of oesophagus, *356.
Staphylococcal infection. *See* Arthritis, pyogenic; Osteomyelitis.
Steel, stainless, hinged prosthesis of, for arthroplasty of knee, *31.
Sternum, fractures, plating of, 652.
Strontium, radioactive
and the skeleton (*symposium*), 394.
uptake and retention in teeth of rabbits, 156.
Subacromial bursitis, 158.
Subtalar joint. *See* Talo-calcaneal joint.
Surgeons, orthopaedic, Edinburgh pioneers, 393.
Sympathectomy, lumbar, results, 661.
Synovial cysts
of fingers, 399.
See also Ganglia.
Synovioma, malignant, 650.
congenital, of bone, *742.

T

Talipes. *See* Club foot.
Talo-calcanean joint, arthrodesis for fractures of calcaneum, *336.
Tarsus
arthrodesis, 402.
Lambrinudi's
for drop foot due to sciatic nerve injury, 221.
modified, *333.
talo-calcanean, for fractures of calcaneum, *336.
See also Calcaneum.
Taylor, Sir Gordon Gordon-. *See* Gordon-Taylor, Sir Gordon.
Teeth
enamel of, organic matrix
in some diseases, 854.
quantitative changes during growth, 853.
radiation damage in rabbits after single injection of strontium-90, 395.
strontium-90 uptake and retention in rabbits, 156.
Tendo calcaneus
anatomy in rabbit, *360.
rupture, etiology, 659.
Tendons
implantation into bone, reaction of bone and tendon after, *377.
See also names of tendons, and under Hand.
Tennis elbow, resection of synovial membrane for, 400.
Tenosynovitis, tuberculous, of calf, 400.
Tetracyclines in measurement of bone growth, 856.
Texas Institute for Rehabilitation and Research, 851.
Thomas's splint in treatment of fractures of femoral shaft associated with head injuries, 425.
Thompson's quadricepsplasty, 159.
Thompson's Vitallium arthroplasty of hip for fractures of femoral neck, 857.
Thorax. *See* Chest.
Thumb
carpo-metacarpal joint of, osteoarthritis, excision of trapezium for, *502.
reconstruction, *444.
Thymidine, tritiated, in autoradiographic study of cell division in endochondral ossification, *824, 857.
Thyroid gland. *See* Hashimoto's disease.
Tibia
fibroma, chondromyxoid, *542.

* Indicates full article or appreciable part.

Tibia
fractures
compound, treatment, comparative study, 407.
stress, longitudinal, *508.
pseudarthrosis, congenital, treatment, 404.
Tibialis posterior transplantation for drop foot, 410.
due to sciatic nerve injury, 224.
Tissue viability, assessment of, intravenous dye technique, 646.
Toes
big
sesamoid bones of
degeneration associated with hallux valgus or hallux rigidus, 158.
disorders, *840.
See also Hallux rigidus; Hallux valgus.
claw
amputation of all toes for, *90; (*correspondence*), 412.
due to sciatic nerve injury, 224.
reconstruction of forefoot for, 399; (*correspondence*), 412.
Trapezium, excision for osteoarthritis of carpo-metacarpal joint of thumb, *502.
Travelling Fellows in Orthopaedic Surgery, British, visit to America, 1960, 847.
Tritiated thymidine in autoradiographic study of cell division in endochondral ossification, *824, 857.
Tuberculosis
bone and joint, in the Bantu, problems of, 861.
hip, 399.
knee, 409.
spine, growth of spine in, and its mechanical control, 47.
Tuberculous tenosynovitis of calf, 400.
Tumours
giant-cell, of bone. *See* Osteoclastoma.
See also other more specific terms, and under names of tissues etc.

U

Ulna
fractures
greenstick, of upper end, with lateral dislocation of radio-humeral joint, *782.
Ulna
fractures
Monteggia, with unreduced dislocation of radial head, 406.
head of, dislocation. *See* Radio-ulnar joint, inferior, dislocation.
Ulnar dimelia, *549.
Ulnar nerve
and median nerve injuries at wrist, repair of, 647.
lesion in javelin thrower, 788.
lesions in fractures of medial epicondyle of humerus, 780.
United States, orthopaedics in, report of Travelling Fellows, 1960, 847.
Urticaria pigmentosa, bone involvement in, 641.

V

Vascular disorders
occlusive, bone vascular reaction in, *110.
treatment, sympathectomy, lumbar, results, 661.
See also Arteries, injuries; Ischaemia.
Vascular tumours. *See* Glomus tumour; Haemangioma; Haemangiopericytoma.
Vertebral column. *See* Spine.
Vitamin D, osteosclerosis due to intermittent administration of large doses in rat, *606.

W

Watson-Jones Lecture, Royal College of Surgeons of England, 1959, 153.
Wrist
arthrodesis in reconstructive surgery, *60.
dislocation, 400.
nerve injuries at, median and ulnar, repair of, 647.
See also Radio-ulnar joint, inferior; Carpo-metacarpal joints; *and names of carpal bones.*
See also De Quervain's disease.

Y

Yaws, gummatous, tumoural, *785.

* Indicates full article or appreciable part.